A Dissertation on

INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL

Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

with partial fulfillment of the regulations

for the award of the degree of

M.S. Degree in General surgery

COIMBATORE MEDICAL COLLEGE

COIMBATORE

APRIL 2016

CERTIFICATE

This is to certify that the dissertation titled **"INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL"** submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch-1 (General Surgery) is a Bonafide work done by **Dr. C. VINODINI,** A post graduate student in General Surgery under my direct supervision and guidance during the period of July-2014 to August-2015.

Prof.Dr.V.Elango, M.S
Professor and Head of the Department
Dept. of General Surgery
Coimbatore Medical College & Hospital
Coimbatore.

Dr. EDWIN JOE, M.D.,B.L.,
The Dean
Coimbatore Medical College Hospital
Coimbatore.

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : Dr. C. Vinodini

Course : MS General Surgery

Period of Study : 2013 - 2016

College : Coimbatore Medical College

Dissertation Topic : Incidence of asymptomatic peripheral artery disease in type 2 diabetes in a tertiary care hospital.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

DEAN
Coimbatore Medical College & Hospital,
Coimbatore

16.7.2014

turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	221311316.m.s.general Surgery Dr....
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	INCIDENCE OF ASYMPTOMATIC P...
File name:	Thesis.docx
File size:	72.1K
Page count:	78
Word count:	7,409
Character count:	44,230
Submission date:	24-Sep-2015 03:56PM
Submission ID:	571425531

A Dissertation on

INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE2 DIABETES IN A TERTIARY CARE HOSPITAL

COIMBATORE MEDICAL COLLEGE
COIMBATORE

A Dissertation on

INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL

Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

with partial fulfillment of the regulations

for the award of the degree of

M.S. Degree in General surgery

DECLARATION

I hereby declare that the dissertation entitled **"INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL"** was done by me at Coimbatore Medical College Hospital, Coimbatore - 18 during the period of my post graduate study for M.S., Degree Branch-1 (General Surgery) from 2014 to 2015.

This dissertation is submitted to the Dr. M.G.R. Medical University in partial fulfilment for the award of M.S., Degree in General Surgery.

Date :

Place :

Dr. C. VINODINI
Post Graduate Student,
M.S., General Surgery,
Coimbatore Medical College & Hospital
Coimbatore.

ACKNOWLEDGMENTS

I am thankful to the **Almighty** for giving me strength to complete this work.

I express my gratitude to **Dr.Edwin Joe, MD.,** Dean, Coimbatore Medical College and Hospital, for providing facilities to carry out this work.

I am thankful to **Dr. Rewathy, MD.,** former Dean Coimbatore Medical College and Hospital for giving me permission to do this work.

I am extremely indebted to **Dr.V.Elango,MS**. Professor and HOD, Department of Surgery, and my guide for showing me the path throughout my work.

I am deeply indebted to **Dr.E.Suresh, MD, D Diab**. Former HOD of Diabetology Coimbatore Medical College; **Dr.S.Vengo Jeyaprasad, MD, D Diab** HOD, Diabetology, Coimbatore Medical College and **Dr.Venkadesh, D Diab,** Tutor Diabetology, Coimbatore Medical College for their help and guidance in each step of the study.

I am thankful to our Professors **Dr.D.N.Renganathan, MS, Dr.S.Natarajan, MS, Dr.G.Raveendran, MS, Dr.S.Sarada MS** and **Dr.S.Balasubramaniyan, MS.**

I express my sincere gratitude to my unit Assistant Professors **Dr.R.Narayanamoorthy, MS; Dr.P.Sumithra, MS, DGO; Dr.Jayalakshmi, MS; Dr.N.Tamilselvan, MS; Dr.T.Srinivasan, MS** and **Dr.R.Radhika MS, DGO.**

Last but not least, I sincerely express ,my gratitude to all my patients, who cooperated with me in this study, making it a success.

LIST OF CONTENTS

LIST OF ABBREVIATIONS

ABI	-	Ankle Brachial index
ATP	-	Adenosine Triphosphate
CHD	-	Coronary heart disease
DAG	-	Diacyl glycerol
DM	-	Diabetes Mellitus
DNA	-	Deoxy Ribonucleic acid
ECG	-	Electro cardio gram
HDL	-	High density lipoprotein
HNF	-	Hepatocyte nuclear transcription factor
IFT	-	Impaired fasting glucose
IGT	-	Impaired fasting glucose
LDL	-	Low density lipoprotein
MODY	-	Maturity Onset Diabetes of Young.
NS	-	Non smoker
PAD	-	Peripheral artery disease
PKC	-	Protein kinase C
PS	-	Passive smoker
RFT	-	Renal function test
UKPDS	-	United kingdom prospective diabetes study

INTRODUCTION

ASYMPTOMATIC PERIPHERAL ARTERY DISEASE

Def: asymptomatic peripheral artery disease is defined as ankle brachial index less than 0.9 in patients with no clinical evidence of peripheral arterial disease or foot ulcer[1]. Ankle brachial index less than 0.9 has got 90% sensitivity and specificity. Low brachial index is a predictor of future myocardial events stroke and amputation. Though prevalence of peripheral arterial disease is high in diabetes, studies looking into the presence of asymptomatic peripheral arterial disease in diabetic patients are very few.

In spite of the understanding that asymptomatic peripheral arterial disease is a very important risk factor for all types of future vascular events including cerebrovascular disease, coronary artery disease and critical foot ischemia which eventually leads to amputations, the interest towards studying such a disease seems to be minimal.

Ankle brachial index is a simple inexpensive diagnostic test used for diagnosis of peripheral arterial disease. Sensitivity is especially low in the elderly. Blood Pressure is measured using sphygmomanometer and

hand held Doppler and the ratio of Ankle Blood Pressure to Brachial Blood Pressure is calculated[2].

Epidemiological studies have demonstrated that patients with peripheral arterial diseases have poor survival when compared to the general population[3]. Diabetic patients with peripheral arterial diseases have even more poor prognosis compared to non-diabetic peripheral arterial diseases[4].

Prolonged duration of diabetes, associated diseases like hypertension, kidney diseases, lipid abnormalities etc also seem to increase the atherosclerotic risk and hence peripheral arterial diseases. Smoking is an important risk factor for all vascular events. In peripheral arterial disease also, smoking forms an important risk factor for its development. Presence of other vascular diseases in any patient is indicative of atherosclerotic disease in the patient and hence possibility of peripheral artery disease in such patients is high.

This study is aimed at finding out the presence of asymptomatic peripheral arterial disease in diabetic individuals and comparing it with non diabetic population.

AIM OF THE STUDY

The aim of the study was to establish the high prevalence of undetected, asymptomatic peripheral arterial disease in type 2 diabetes in patients attending Coimbatore Medical College and Hospital.

OBJECTIVES

1. To establish the hypothesis that asymptomatic peripheral artery disease is common in diabetes.
2. To assess the relation of asymptomatic peripheral arterial disease to the sex of the patient.
3. To establish passive smoking as an additional risk factor.
4. Risk identification and outcome prediction, thereby preventing complications like amputations.

MATERIALS AND METHODS

Study design

This was a cross sectional, case- control study of patients attending Coimbatore Medical College with (cases) or without (controls) Diabetes

Time period

August 2014 to July 2015

Inclusion criteria

100 diabetic patients without clinical evidence of peripheral vascular disease or other vascular diseases and an equal number of non diabetics attending hospital for other ailments were included as cases and controls respectively.

Exclusion criteria

1. Established atherosclerotic diseases like coronary artery disease, stroke or peripheral vascular diseases.
2. Smokers.
3. Patients with leg claudication or chronic leg pain syndrome.
4. Patients with absent lower limb pulses

5. Bed-ridden patients.

6. Diabetic foot ulcer patients.

Data collection methods

- Informed consent was taken from all the participants prior to examination.

- Detailed history including duration of diabetes, vascular diseases, dyslipidaemia, smoking, alcohol use and drugs were noted.

- A thorough physical examination including all peripheral pulses and carotid pulses were examined.

- Blood pressure measured using aneroid/ digital sphygmomanometer using a stethoscope.

- Brachial and ankle systolic blood pressures were measured using a hand held Doppler.

- Routine investigations including blood sugars, lipids, RFT, and ECG were done.

- Data recorded in a proforma, tabulated and statistically analyzed using online statistical tools.

REVIEW OF LITERATURE

Diabetes Mellitus:

Definition: Diabetes mellitus is defined as a heterogeneous group of diseases characterized by hyperglycemia secondary to increased production of glucose and / or decreased utilization or both.

Diabetes mellitus refers to a group of disorders presenting with hyperglycemia, having several distinct causes, resulting from complex interactions of genetic and environmental factors. Hyperglycemia results from decreased insulin secretion decreased glucose utilization or increased glucose production[5].

Classification[6]

1. Type 1 Diabetes (insulin deficiency)

   A-immune mediated B-Idiopathic

2. Type 2 Diabetes (insulin deficiency,relative insulin deficiency-insulin secretory defect)

3. Other specific types of diabetes

A. Genetic defects of B cell function characterized by mutation in

a. Hepatocyte nuclear transcription factor (HNF) 4 alpha (MODY 1)

b. Glucokinase (MODY 2)

c. HNF 1 alpha (MODY 3)

d. Insulin Promoter Factor 1 (MODY 4)

e. HNF 1 b (MODY 5)

f. Neuro D1(MODY 6)

g. Mitochondrial DNA

h. Subunits of ATP sensitive potassium channel

i. Proinsulin or Insulin

B. Genetic defects in insulin action

a. Type A insulin resistance

b. Leprechaunism

c. Rabson Mendenhall syndrome

d. Lipodystrophy syndromes

C. Diseases of exocrine pancreas

a. Pancreatitis

b. Pancreatectomy

c. neoplasia

d. cystic fibrosis

e. haemochromatosis

f. fibrocalculous pancreatopathy

g. mutations in carboxyl esterlipase

D. Endocrinopathies:

a. acromegaly

b. cushings syndrome

c. glucagonoma

d. pheochromocytoma

e. hyperthyroidism

f. somatostatinoma

g. aldosteronoma

E. Drug or chemical induced

a. glucocorticoids

b. rodenticide –vacor

c. pentamidine

d. nicotinic acid

e. diazoxide

f. beta-adrenergic agonists

g. thiazides

h. hydantoins

i. asparginase

j. alpha-interferon

k. protease inhibitors

l. antipsychotics (atypical and others)

m. epinephrine

F. Infections

a. congenital rubella

b. cytomegalovirus

c. coxsackie virus

G. Uncommon forms of immune mediated diabetes

a. stiff person syndrome

b. anti insulin receptor antibodies

H. Other genetic syndromes

a. wolfian syndrome

b. down syndrome

c. klinfelters syndrome

d. turners syndrome

e. freidreichs ataxia

f. huntingtons chorea

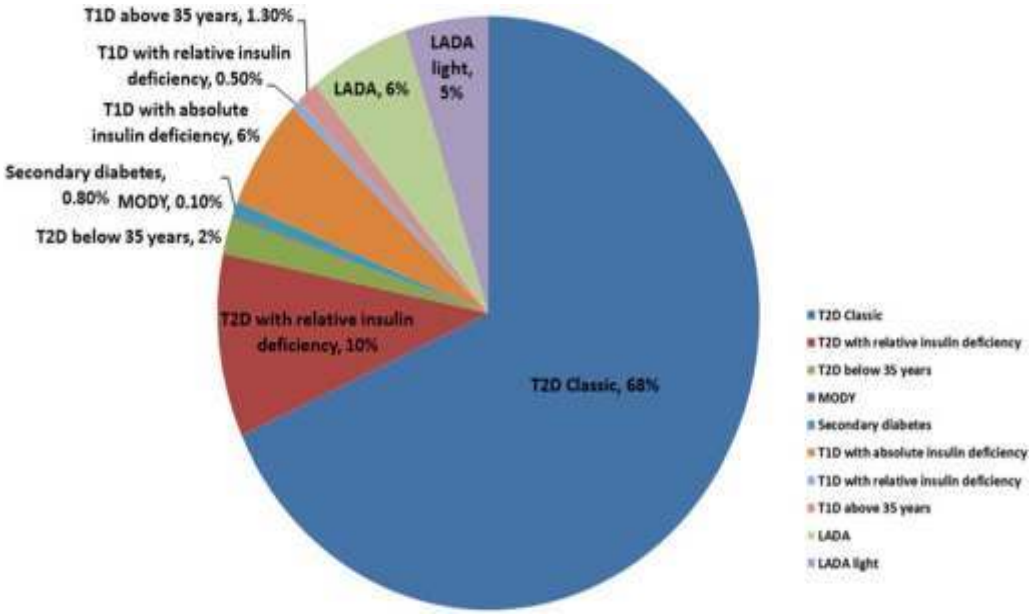
g. Laurence moon biedl syndrome

h. mytonic dystrophy

i. porphyria

j. Prader willi syndrome

4. Gestational Diabetes Mellitus

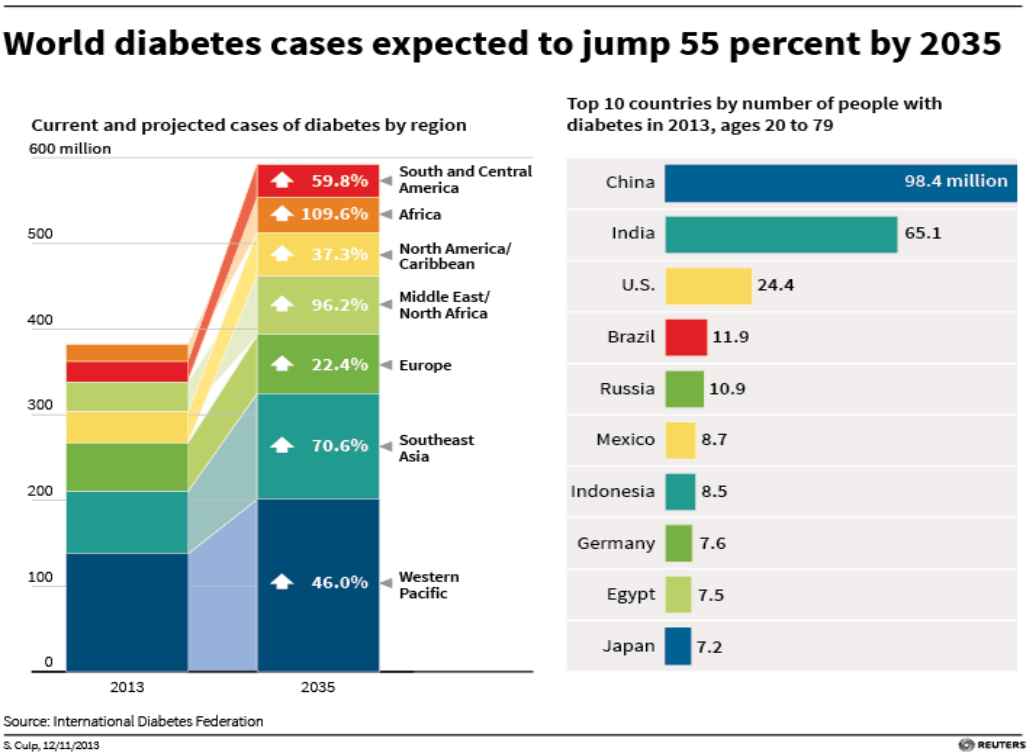


Type2 Diabetes Mellitus

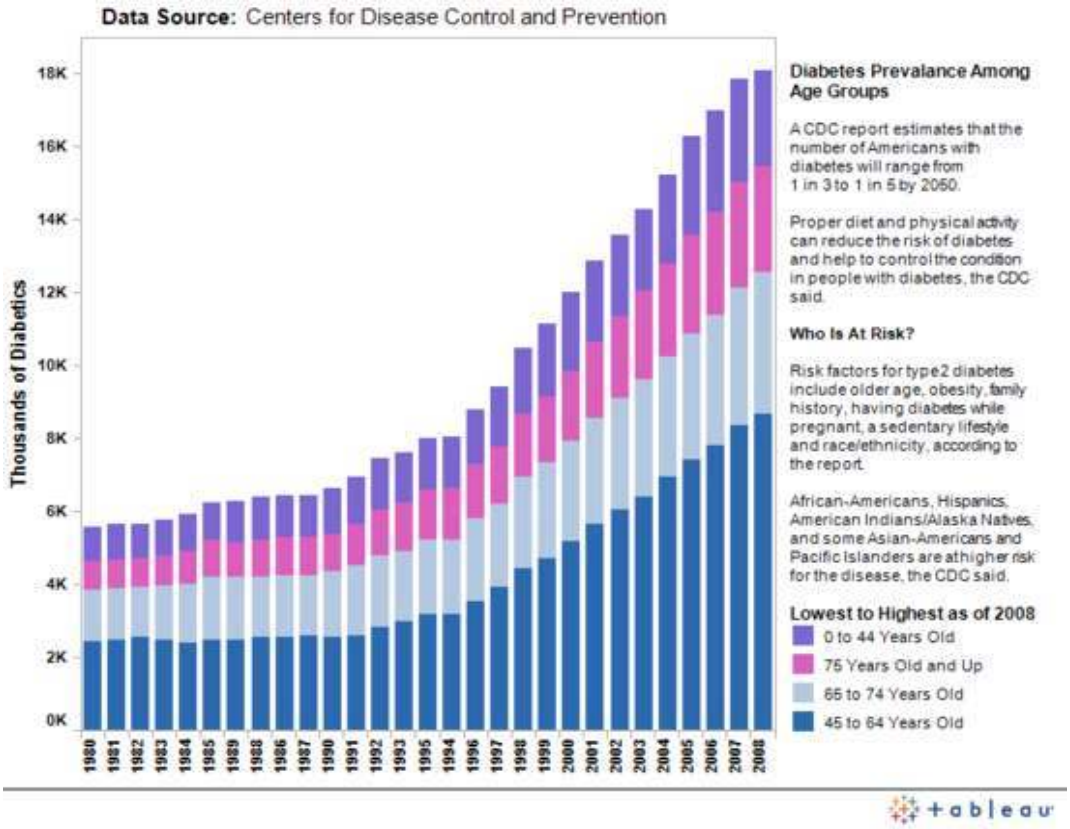
Type2 Diabetes is a heterogeneous group characterized by combination of insulin resistance and insulin secretary defect associated usually with increased insulin production initially. Type2 Diabetes may be preceded by a period of abnormal glucose homeostasis manifested as a raised fasting blood sugar (Impaired Fasting Glucose- IFG) or an increased post meal (glucose) blood sugar (Impaired Glucose Tolerance- IGT)[5].

Type2 Diabetes is ever increasing worldwide and in India, and more so in commonly South India[7]. By 2025 the global burden of type2 Diabetes was estimated to rise to 270 million patients, an alarming situation, probably secondary to sedentary life style and resultant obesity

as per a 1998 study[8]. By 2010 Diabetes cases worldwide (type1, type2 and others together) was estimated at about 285 million and International Diabetes Federation projects that 438 million people worldwide will be affected by Diabetes by 2030, of which, majority of course will be type2 diabetes[6].

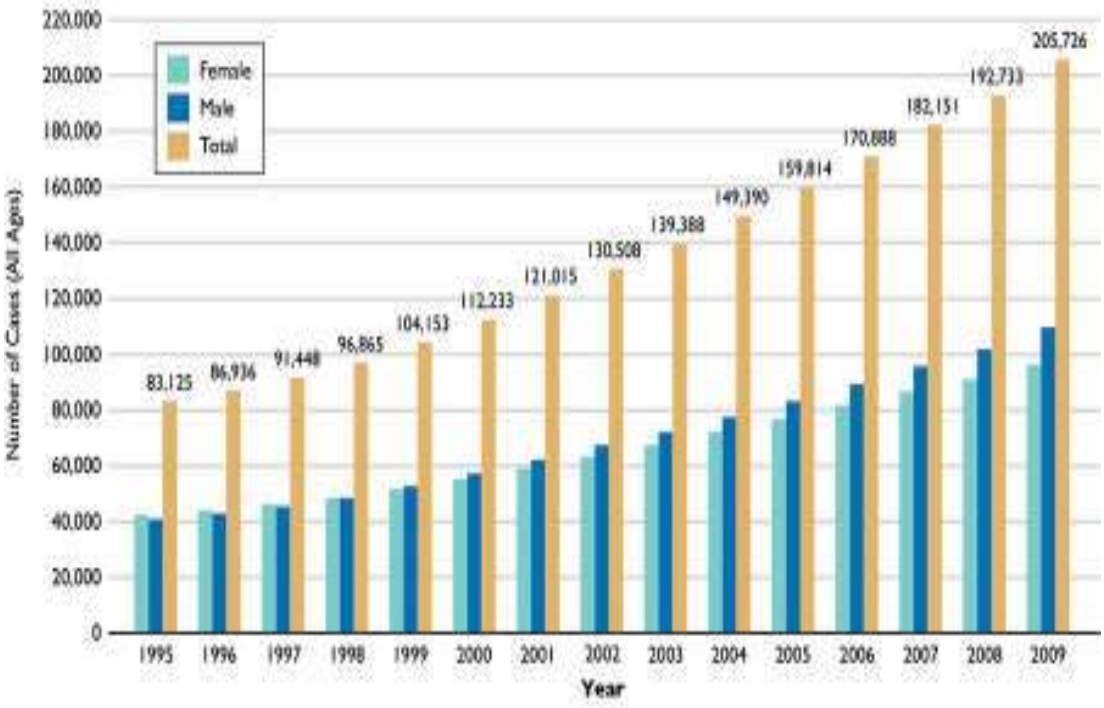


More and younger individuals are affected by diabetes and people on their middle age are most affected by diabetes. Reason for a shift to left of type2 diabetes is probably sedentary lifestyle and increasing indulgence in junk foods. The fact that more diabetics survive for longer periods results in more older diabetics and more people with prolonged diabetes and hence more of complications.



Both males and females are equally affected by diabetes, and the numbers are increasing in both sexes over time. However the increase is more profound in males compared to females.

Figure 2.1 **Prevalent Diabetes Cases, 1995-2009**



The burden of diabetes is not merely the number of diabetic cases, but the enormous cost involved in the treatment of diabetes and its complications. More over diabetic foot diseases result in significant morbidity and mortality both by itself and by associated coronary artery disease and cerebrovascular disease.

Diagnostic criteria for diabetes mellitus

1) $HbA_1C > 6.5\%$

   (Tested using a method i.e) National Glyco hemoglobin standardization Programme – Certified and standardized)

2) Fasting plasma glucose level > 126 mg/dl (7.0 mmol/lit)

3) 2 hr. post glucose plasma glucose level > 200 mg/dl (11.1 mmol/lit)

4) In patients with classic symptoms a random plasma glucose level > 200 mg/dl (11.1 mmol/lit)

Prediabetes

A fasting plasma glucose level 100-126 mg/dl is known as impaired fasting plasma glucose. (IFG) and 2 hr plasma glucose 140-200 mg/dl is known as impaired glucose tolerance (IGT).

IFG & IGT are together called prediabetes. People with prediabetes have similar vascular and atherosclerotic risks as in diabetics.

Treatment of Diabetes : [5,6]

The aim of treatment of diabetes mellitus is to alleviate symptoms and to prevent or atleast slow down complications.

Glycemic control and blood pressure control are mainstay in control of microvascular complications (retinopathy, nephropathy). In prevention of macrovascular complications (coronary artery disease, peripheral vascular disease and cerebrovascular disease) aspirin therapy, smoking cessation and lipid control also play a major role in addition to

glycemic control and hypertension control. Glycemic control is important in preventing other metabolic complications also.

Diabetic treatment consists of many interventions integrated. Nutritional interventions, promoting physical activity, smoking cessation, pshychosocial care, glycemic treatment, setting individual therapeutic goals, health education, early detection and treatment of complications and intensification of insulin therapy in type 2 diabetes mellitus. Ideal diabetic care is done by a multidisciplinary team comprising of diabetic physician, diabetic educatior, nutriontist, diabetic nurses, ophthalmologist, cardiologist, nephrologist, neurologist, podiatrician, and social workers.

Ideal treatment target in diabetes is to maintain pre-prandial Blood sugar = 90 – 130 mg/dl and HbA1C < 7.00 percent.

Diabetic treatment goals are not fixed and are dependent on other morbidities, risk profile of the patient and life expectancy of the patient.

Aggressive glycemic treatment goals are not advisable for elderly patients, those with advanced malignancies, those with extensive coronary artery disease etc.

Whereas an young patient with no other co-morbidities can have a aggressive management goals as tight as HbA_1C 6%

Early treatment of diabetes mellitus can lead to longterm positive benefits and uncontrolled diabetes in the early stages can relentlessly progress to complications inspite of better glycemic control at later stage (Metabolic memory).

Non pharmacologic treatment

In many patients with diabetes intial intervention to be offered is lifestyle modification. Lifestyle modifications include improvements in physical activity, therapeutic dietary interventions and smoking cessation.

Dietary modification

It is of atmost importance to note that dietary interventions does not mean imposing undesirable and unpalatable foods which patients are unlikely to follow. Ideal dietary intervention should be based on locally available foods which suites the culture and customs of the patient.

Caloric restriction is most important. Depending on the patients physical activity and actual body weight versus ideal body weight, calorie requirements is calculated. Modest restriction of saturated fats and simple sugars is needed. 5-10% weight loss if achieved is associated with

significant improvements in HbA_1C levels, Blood pressure, triglycerides and increase in HDL cholesterol. And hence will result in significant reduction in risk of vascular disease.

10-15% weight reduction can result in even better glycemic control and cardiovascular risk reduction. High protein low carbohydrate diets can result in better sugar control.

Physical activity modification

Increased physical activity in the form of aerobic exercise can improve insulin sensitivity and blood sugar values. Structured exercises of >150 min/week is associated with greater HbA_1C reduction. However physical activity will help to lower blood sugar only when combined with dietary modifications.

Patient should be allowed to choose a physical activity which he or she is likely to continue. eg. walking, jogging, swimming. A previously sedentary patient should not jump into severe physical activity to start with. Rather a gradual start is advisable for older patients, those with long standing diabetes, patients with multiple risk factors and those with previous atherosclerotic diseases should have proper cardiac evaluation prior to starting exercises.

Pharmacologic Therapy

Drugs used in diabetes can be classified as follows.

1) Biguanides
2) Sulfonyl ureas
3) Meglitinide derivatives
4) Alfa-glucosidase inhibitors
5) Thiazolidine diaones
6) Glucogan like peptide-I (GLP-1) agonists
7) Dipeptidyl peptidase IV (DPP4) inhibitors
8) Selective sodium glucose transporter II (SGLT-II) inhibitors
9) Insulins
10) Amylinomimetics
11) Bile acid sequestrants
12) Dopamine agonist

Biguanides

Metformin is the only clinically used drug in this group. Another biguanidees, phenformin was withdrawn from the market due to high incidence of lactic acidosis. Metformin is time tested molecule used as first line therapy of type 2 diabetes. Mechanism of action is by inhibiting hepatic gluconeogensis. It also probably decreases peripheral insulin

resistance. Additional uses of metformin include treatment of polycystic, ovarian disease and treatment of obesity. It is relatively safe and seldom produces hypoglycemia by itself. Significant HbA_1C reduction is seen with metformin especially when the initial HbA_1C is very high. Side effects are diarrhea, flatulence and intestinal pseudo obstruction.

Sulpfonyl ureas

Sulfhonyl ureas are insulin secretagogues that stimulates insulin secretion from pancreatic beta cells. They are highly potent and effective, though the actions may be short lived. There is gradual loss of beta cell function due to apoptosis and hence reduce insulin production. They are used as adjuncts to diet and exercise in type 2 diabetes possibly as add on to metformin. Examples glibenclamide, glipizide, gliclazide and glimipride side effects are hypoglycemia, allergic rashes, steven johnson syndrome etc.

Meglitinide derivatives

Eg. Repaglinide, Nateglinide

They are short acting insulin secretagogues also known as prandial regulators. They also act in a way similar to sulfonylureas but the effect is more physiological and short lived. They are much more

costly when compare to sulfonylureas. Side effects is hypoglycemia much less then sulfonylureas.

Alfa-glucosidase inhibitors

Examples : Acarbose, Voglibose

They act by inhibiting the enzyme alfa-glucosidase which facilitates the breakdown of complex sugars into simple sugars. Thus alfa-glucosidase inhibitors prevents absorbtion of carbohydrates. Troublesome side effect of flatulence limits their use.

Thiazolidine diones

Examples : Pioglitazone, Rosiglitazone

They act on peroxisome proliferator activated receptor γ. Once widely used they have fallen into ill repute and is sparingly used now. Rosiglitzone is already withdrawn from market due to inadequate cardiac safety. There was a hue and cry regarding bladder cancer with the use of pioglitazone and it was temporarily banned from an Indian market.

These drugs are highly effective in reducing insulin resistance and facilitating glucose uptake in peripheral tissues especially adipose tissue. Pioglitazone additionally have beneficial effects in non alcoholic steatohepatitis and favourable lipid regulatory effect. Pioglitazone can be

used as monotheraphy or in combination with metformin, sulfonylureas, meglitinides, DPP4 inhibitors, GLP_1 receptor agonist or insulin. Side effects are fluid retention, weight gain, worsening cardiac failure fractures and probably bladder cancer. Saraglitazar a combined PPAR – α and γ agonist is currently available having favourable effects on triglycerides and glucose.

GLP_1 agonists

Examples. Exenatide, Liraglutide albiglutide, Dulaglutide.

They act by mimicking the endogenous incretin. GLP-I. They stimulate glucose dependent insulin release, reduce glucagon and delay gastric emptying. They promote moderate weight loss. There is animal data suggesting that they prevent beta cell apoptosis and hence restoring beta cell mass, though not proven in humans. Side effects include nausea vomiting and abdominal discomfort.

Liraglutide the long acting GLP-I analogue has been approved by the USF DA for treatment of obesity.

Dipeptidyl Peptidase IV inhibitors

Examples. Sitagliptin, Vildagliptin, Saxagliptin, Linagliptin, Allogliptin

They act by inhibiting the enzyme dipeptidyl peptidase IV which is responsible for degradation of endogenous incretins, thus prolonging incretin effect. They can be used as monotherapy or in combination with metformin, thiazolidinediones and insulin.

They are more or less weight neutural and produce moderate reduction in HbA1C. Upper respiratory tract infections are increasingly reported with the use of DPP-IV inhibitors. Hypoglycemia is seldom seen.

Selective Sodium Glucose transporter II – inhibitors

Examples. Canagliflozin, Dapagliflozin, Empagliflozin

They act by decreasing renal threshold of glucose which result in increased excretion of glucose in urine. They can be used in combination with metformin, sulfonyl ureas, DPP IV inhibitors. Their use is limited when the GFR is low. Increased incidence of urinary tract infection is seen with use of SGLT II inhibitors.

Insulins

Bovine and porcine insulins were used earlier but now recombinant human insulins are in use. Regular Insulin is short acting and

is used as multiple doses for diabetes treatment and can be used as intravenous infusion in emergencies.

NPH insulins are intermediate acting and can be used as twice daily regimens alone or in combination with regular insulin (30/70 or 50/50 premixed combinations are available in market). Insulin analogues are widely used nowadays. There are ultrashort acting insulin analogues, examples Lispro, Aspart and Glulysine and ultralong acting insulin analogues. eg. detrimer, glargine, deglutec available.

All patients of Type I diabetes and most of advanced Type 2 diabetes will require insulin. Acute complications of diabetes, infection, pregnancy, surgeries and renal failure will require insulin therapy. Hypoglycemia is the dreaded side effect of insulin therapy. Weight gain is experienced by most of the patients with insulin therapy.

Amylinomimetics :

Examples : Pramlintide acetate. Amylin is secreted by pancreatic beta cells. It delays gastric emptying increased postprandial glucagon release and causes satiety. Wide clinical experience is lacking with this group.

Bile acid sequestrants

Examples : Colesevelam

Initially developed as a lipid lowering agent subsequently found to have glucose lowering effects. Can be used as a adjunctive therapy to improve glycemic control. Side effects are abdominal discomfort and flatulence.

Dopamine agonists :

Eg) Bromocriptine mesylate

Bromocriptine is a centrally acting D_2 receptor agonist. When given as a short acting formulation in single morning dose they may act on circadian neuronal activities within hypothalamus to reset the abnormally elevated drive for plasma glucose. It do not cause weight gain or hypoglycaemia. Orthostatic hypotension and syncope can be troublesome esp. during initiation of therapy.

Surgical treatment for diabetes – Bariatric surgery

In morbidly obese patients bariatric surgery has shown to improve diabetes control and in some patients even normalizes blood sugar. It is important that the patients are carefully selected for surgery.

Bariatric Surgeries :

I. Restrictive

- Vertical banded gastroplasty
- Laparoscopic adjustable gastric banding (LAGB)
- Jaw wiring

II. Malabsorptive

- Biliopancreatic diversion (BPD)
- Biliopancreatic diversion with duodenal switch (BPD-DS)
- Jejunoileal bypass

III. Combined

IV. Roux-en-Y-gastric bypass (RYGB) open or laparoscopic.

Rou-en-Y-gastric bypass is having best sustained effect on diabetes remission.

Prevention of type 2 diabetes :

Prevention of type 2 diabetes mainly relies on the following Principles

1. Weight reduction
2. Therapeutic Nutritional intervention
3. Regular physical activity

4. Cardiovascular risk reduction
5. Aggressive treatment of hypertension and dyslipdemia

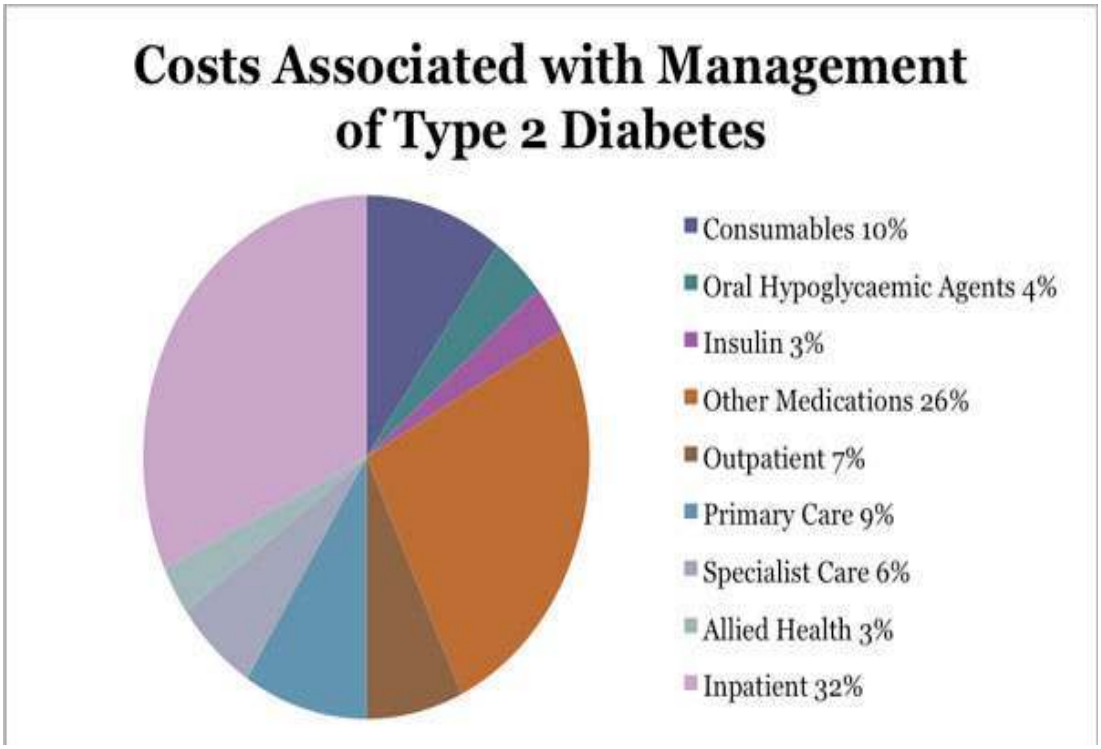
Life style modification

Life style changes with 4-5% sustained weight reduction has been shown to decrease risk for diabetes in patients by 58%.

Life style modification includes caloric restriction, regular physical activity and cessation of smoking etc.

Pharmacologic prevention

Though many drugs are tried, USFDA has not approved any durg for prevention of diabetes. However metformin and pioglitazone are shown to be beneficial in prevention of diabetes.



Peripheral Artery Disease

Peripheral artery disease is a major macro vascular complication of diabetes, often leading to ischemia and subsequent diabetic foot complications including amputations. It is highly prevalent among diabetics, both men and women. Symptoms of peripheral arterial disease include intermittent claudication, rest pain, numbness of extremities, parasthesias, loss of hairs, skin changes like blackish discoloration and gangrenes.

Asymptomatic peripheral artery disease, as evidenced by ankle-brachial index less than 0.9 is also common in diabetic men and women. Most of these patients can, in their later life, develop significant, clinically evident peripheral arterial diseases and its complications. Other vascular diseases are also notably high in these patients. Detection of peripheral arterial disease in an early stage, when asymptomatic, can help the clinician to decide on necessary preventive strategies like strict glycemic control, control of lipids, cessation of smoking, control of hypertension, prevention of foot infections, proper podiatric care etc and can reduce the progression of disease to a stage requiring amputation. Preventive strategies are also important in preventing other vascular diseases like coronary artery disease and cerebrovascular disease.

In a South Indian study, type2 diabetic women had a high prevalence of asymptomatic peripheral arterial disease compared to non-diabetic women[9].

Undetected peripheral arterial disease is fairly common in type 2 Diabetes patients with additional risk factors as evidenced by literature. Traditional risk factors like smoking, age and dyslipidaemia are also relevant in type2 Diabetes and renders increased susceptibility to vascular diseases. Atherosclerosis being a systemic disease, peripheral arterial

disease often coexists with other vascular diseases like coronary artery disease and cerebrovascular diseases, and hence a study of peripheral artery disease is ideally done with a setting where compounding risk factors are excluded. Other forms of vascular diseases are also excluded in view of their co-existence with peripheral arterial disease.

Age is an important determinant of atherosclerotic events. As age advances, all atherosclerotic events also increase. Autopsy evidence shows that atherosclerotic process in blood vessels starts as early as at the age of two years. It then progresses relentlessly to end in various forms of vascular events.

Atherosclerosis is no longer a disease of the rich. It is common across all socioeconomic strata. There is very little difference between the rich, the middle class and the poor.

Alcohol use cause some changes in lipids like an increase in HDL, which may help in decreasing the vascular events. It is important to remember that use of alcohol in moderation only will be helpful; which is seldom possible with most of the individuals.

Non Invasive Diagnostic Evaluation- Ankle-Brachial Index (ABI)

ABI is more and more used in evaluation of patients at risk of cardiovascular diseases. A lowABI (less than 0.9) is associated with increased risk of coronary events and indicates significant, even though asymptomatic, underlying peripheral vascular disease.

Measuring ABI[10]:

Blood pressure is measured in both upper limbs and the highest systolic pressure is taken as denominator. The ankle pressure is measured by keeping BP cuff above the ankle, assessing the return to flow of the dorsalis pedis and posterior tibial arteries using a pencil Doppler probe over each artery. Thus measured ankle pressure becomes the numerator. The ratio of ankle pressure to highest arm systolic pressure estimates the ankle brachial index (ABI), normal being more than 1. Patients with intermittant claudication usually have an ABI in the range of 0.5-0.7 and those with rest pain are in the range 0.3- 0.5. An ABI less than 0.3 is usually noted in those with gangrene. These values can change, depending on the degree of compressibility of the vessel wall. The test is having little value in heavily calcified vessel. Due to non-compressibility of blood vessel as certain cases of diabetes and end stage renal disease can have ABI of 1.4 or more and will require additional tests to evaluate

Peripheral Arterial Disease (PAD). Alternate tests include toe-brachial pressure, pulse volume recordings, transcutaneous oxygen measurements or vascular imaging (duplex ultrasound).

$$ABI = \frac{\text{Higher of the ankle systolic pressure}}{\text{Higher arm systolic pressure}}$$

Ankle brachial index has a 90%sensitivity and specificity in identifying peripheral artery disease when value less than 0.9 is taken as diagnostic.[11]

Possible errors in measurement of ABI

1. Observer error: Reading can change from person to person, especially so in inexperienced hands. This can be overcome by experienced physician / technician doing the test.

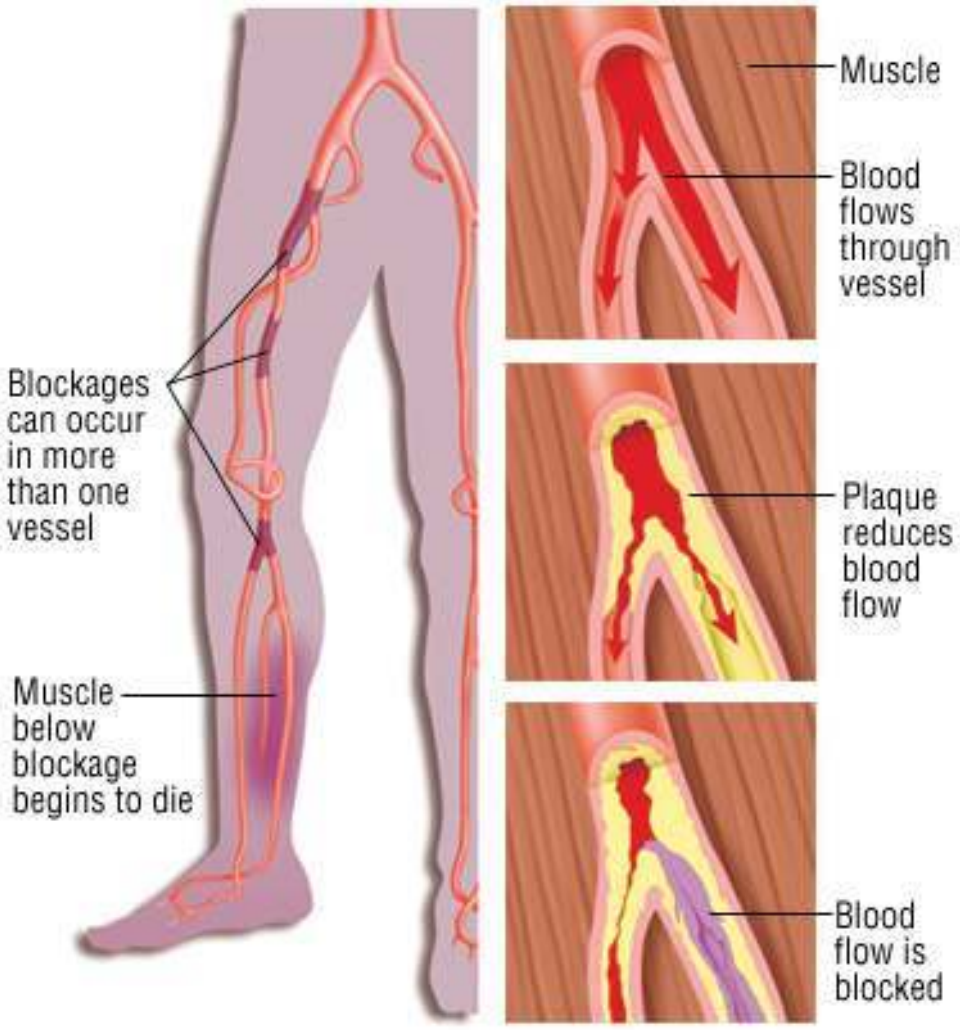
2. Instrument related errors: There can be minor variations in the measured ABP depending on the quality of instrument used. Properly standardized good quality instruments can avoid this error.

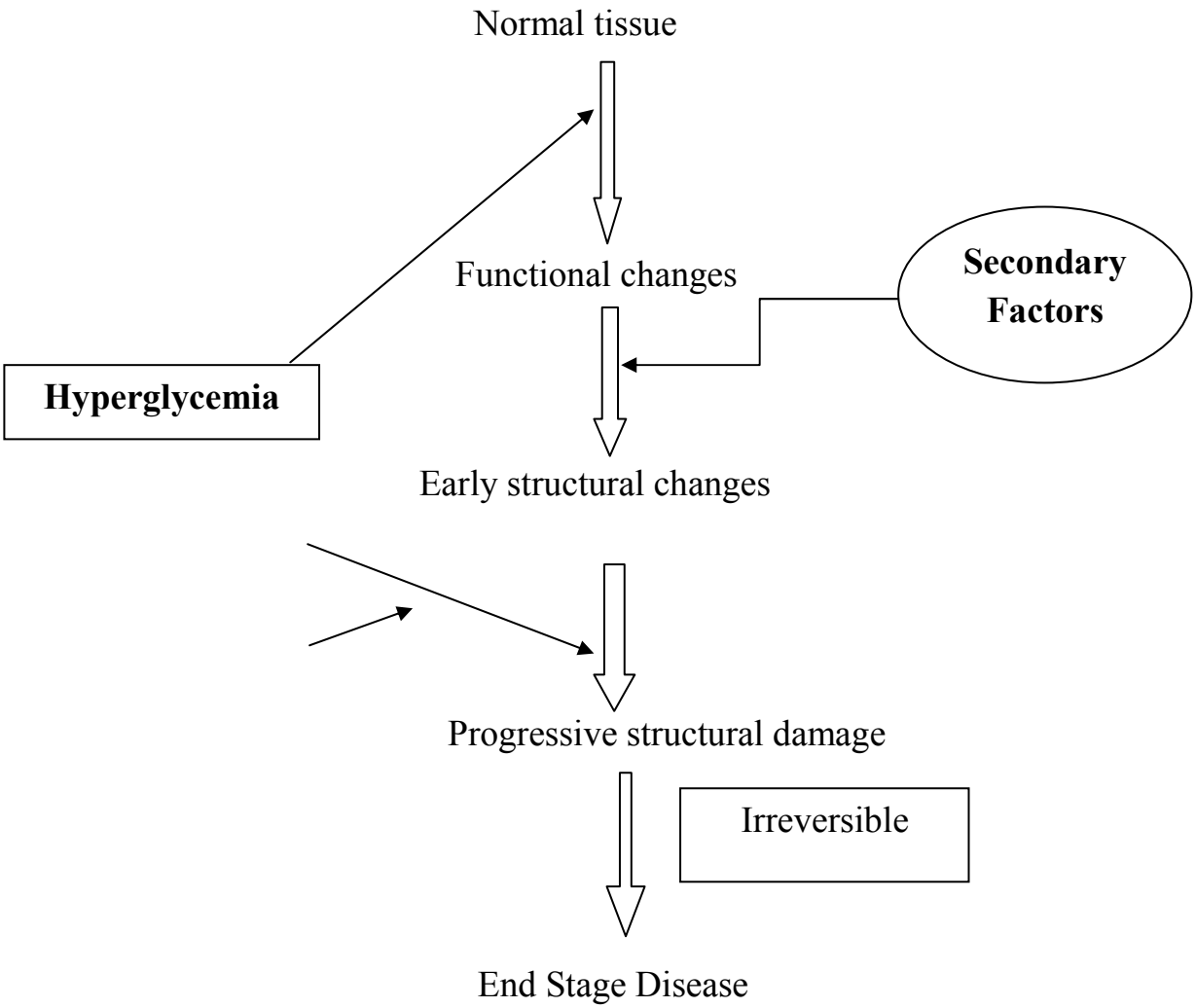
3. Patient related errors like certain drugs, cold exposure, smoking, stress, physical factors etc should be properly evaluated and excluded.

4. Environmental factors: All tests needs to be done in similar environmental conditions to avoid any possible errors.

Pathogenesis of Peripheral Artery Disease in Diabetes[12]

Pathogenesis of chronic diabetic complications is a process that occurs in stages over a period of years. Hyperglycemia remains the core the issue with secondary contributory factors like hypertension, hyperlipedaemia, smoking, alcohol use, dietary excess or deficiency or other environmental toxins. Once structural changes set in further progression to end stage disease is independent of hyperglycemia and there could be some genetic determinants also.


Blockages can occur in more than one vessel
Muscle below blockage begins to die
Muscle
Blood flows through vessel
Plaque reduces blood flow
Blood flow is blocked


Normal tissue
Functional changes
Secondary Factors
Hyperglycemia
Early structural changes
Progressive structural damage
Irreversible
End Stage Disease

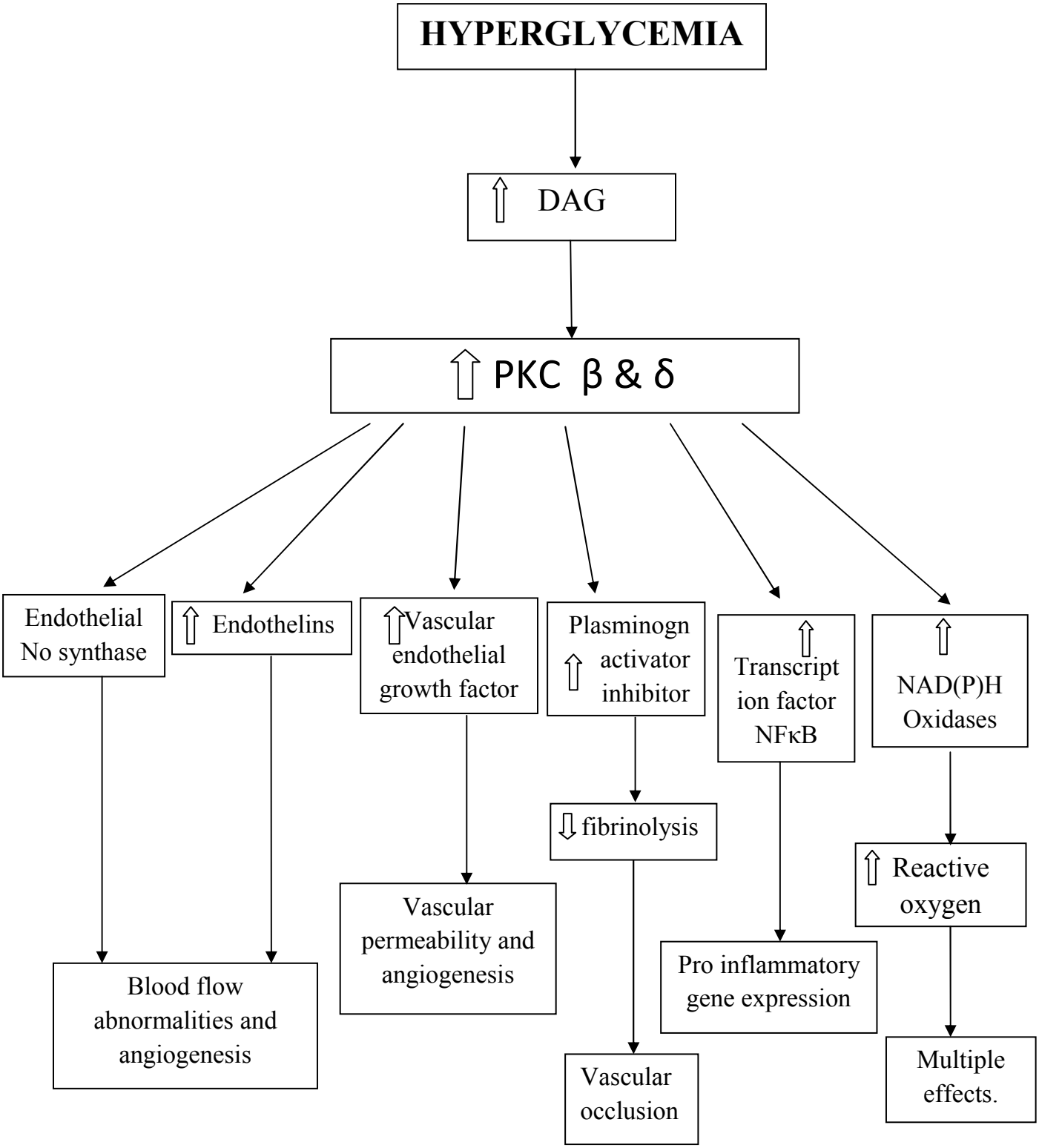
Hyperglycemia can result in tissue damage through various pathways[13]:

1. Increased Polyol pathway flux- resulting in formation of polyalcohol, sorbitol by enzyme aldose reductase. The percentage of this pathway in hyperglycemia varies from species to species and site to site. Normally sorbitol is converted to fructose by sorbitol dehydrogenase. But in hyperglycemia, when sorbitol levels are high, conversion to fructose becomes ineffective and accumulation of sorbitol results in osmotic stress. Aldose reductase inhibitors like tolrestat and epalrestat act by inhibiting the enzyme aldose reductase, thereby reducing sorbitol production.

2. Increased intracellular Advanced Glycation End products. Advanced glycation end products are found in extracellular structures of Diabetic retinal vessels and renal glomeruli. They probably arise secondary to intracellular hyperglycemia, leading to formation of reactive carbonyls(methyl glyoxal and glyoxal) which combines with extracellular proteins. These advanced glycation end products thus formed will alter intracellular protein function, interfere with normal matrix-matrix and matrix and

matrix-cell interactions and also results in pathological changes gene expression, thus resulting in tissue damage.

3. Activation of Protein kinase C: Intracellular hyperglycemia results in increased levels of di-acyl glycerol (DAG), formed from glycolysis intermediate, glyceraldehydes-3- phosphate. DAG activates protein kinase C (PKC), especially the β and δ isoforms.


HYPERGLYCEMIA
⇧ DAG
⇧ PKC β & δ
Endothelial No synthase
⇧ Endothelins
⇧Vascular endothelial growth factor
Plasminogn ⇧ activator inhibitor
⇧ Transcript ion factor NFκB
⇧ NAD(P)H Oxidases
⇩ fibrinolysis
⇧ Reactive oxygen
Blood flow abnormalities and angiogenesis
Vascular permeability and angiogenesis
Pro inflammatory gene expression
Vascular occlusion
Multiple effects.

All these mechanisms will result in changes that will finally result in chronic complications, both micro and macro vascular.

Vascular changes include changes in flow rate, changes in vascular permeability, endothelial dysfunction, pro-coagulability and degeneration.

Dilatation

Growth inhibitors

Anti thrombosi

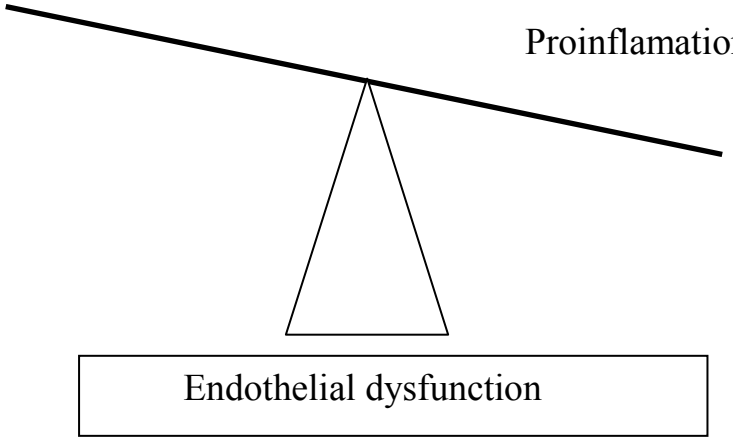
Growth promotion

Constriction

Anti inflammation

Prothrombosis

Proinflamation



Lipid abnormalities in Diabetes[14]

Being an anabolic hormone, insulin promotes esterification of fatty acids, uptake of glucose in adipose tissue as well as liver and muscles, and conversion of glucose to glycogen. Hormone sensitive lipase activity in adipose tissue is inhibited by insulin. In insulin deficient states, there is extensive mobilization of fatty acids from adipose tissue and lipolysis which leads to ketone formation and ketoacidosis.

UKPDS (United Kingdom Prospective Diabetes study has shown that hypertriglyceridaemia is already present at the time of diagnosis of type2 diabetes[15]. This may be caused by additional factors like hypothyroidism, obesity and other genetically determined lipid abnormalities. Type2 diabetes is also characterized by a low HDL. Coronary morbidity and mortality are high when cholesterol levels are higher in both diabetic and non-diabetic individuals, especially the nonHDL cholesterol[16]. But at similar levels of cholesterol and LDL, diabetics are at a greater risk of vascular disease, probably due to qualitative changes in lipoproteins. Diabetics have a greater glycation of their LDL. Diabetics also seem to have more of small LDL which seems to be more atherogenic.

Dyslipidaemia in diabetes is closely correlated to the glycaemic control. The characteristic lipid abnormality, namely hyper triglyceridaemia often decreases with glycaemic control.

Abnormal lipids contribute in a major way in the development of vascular diseases in diabetes including peripheral vascular diseases, their progression, development of complications and outcome.

Peripheral arterial disease in Diabetes

Peripheral artery disease is an important health care problem due to high incidence and prevalence as well as its complications. A number of clinical and epidemiological studies have shown the association of cumulative peripheral arterial disease incidence with duration of diabetes and patients age. There include Framingham (n = 4317)[17,18], UGDP (n= 619)[19], Rochester (n=1703)[20], Kristian stand (n=374)[21], Munchen (n=623)[22], Oxford (n=186)[23], Pittsburg (n=657)[24] and Zagreh (n=200)[25].

STUDY	INCIDENCE	PREVALENCE
Framingham (n=4317)	Claudication M=12.6/1000yrs F= 8.4/1000yrs	18.8 (diabetes duration 16 yrs)
UGDP(n=619)		Palpation M=34.5% F=37.6% Claudication M=37.7% F=24.3% Diabetes duration 20yrs
Rochester (n=1073)	Pulse M=21.3/1000yrs F= 17.6/1000yrs	15% (diabetic duration 10 yrs) 45%(diabetic duration 20yrs)
Kristian stand		Pulse 16.4 (DM duration 1.5 yr) 38.7(DM duration 2yrs)
Munchen (n=623)		Ultrasound M=18.0% F=14.4%
Oxford(n=186)	Claudication 16/1000yrs	
Pittsburg (n=657)		M=11.0% F>30% (DM duration >30 yrs)
Zagreb(n= 200)		Plethysmography :15% DM duration % yrs 18.5% DM duration 10 yrs

Prevention of peripheral vascular disease

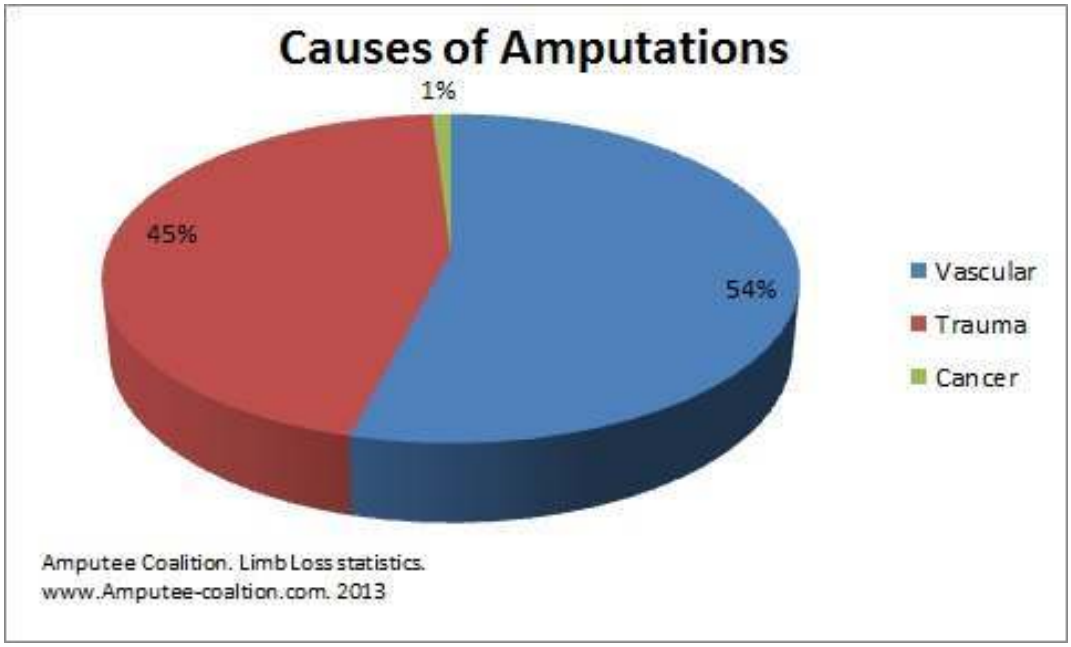
Glycemic control is the most important intervention in prevention of peripheral vascular – disease as well as coronary vascular diseases and cerebral vascular diseases.

Other pharmacologic intervention include aspirin, statins and ACE inhibitors.

Amputations

Diabetes is the leading cause of non-traumatic amputation of extremities in the world. About 1.8 million Americans are living with amputations. Reasons for foot complications in Diabetes include neuropathy, pressure ulcers, skin changes and ischemia secondary to peripheral arterial disease.

Most literature reveals that vascular causes account for majority of amputations are due to vascular causes followed by trauma. Malignancy forms a minor cause of amputations. Arterial disease is major vascular reason for amputations.



Disease wise cause of amputations in literature puts diabetes as the major cause followed distantly by trauma.

Aims of aimputation

1) To excise all pathology
2) To restore the all maximal limb function

Indication

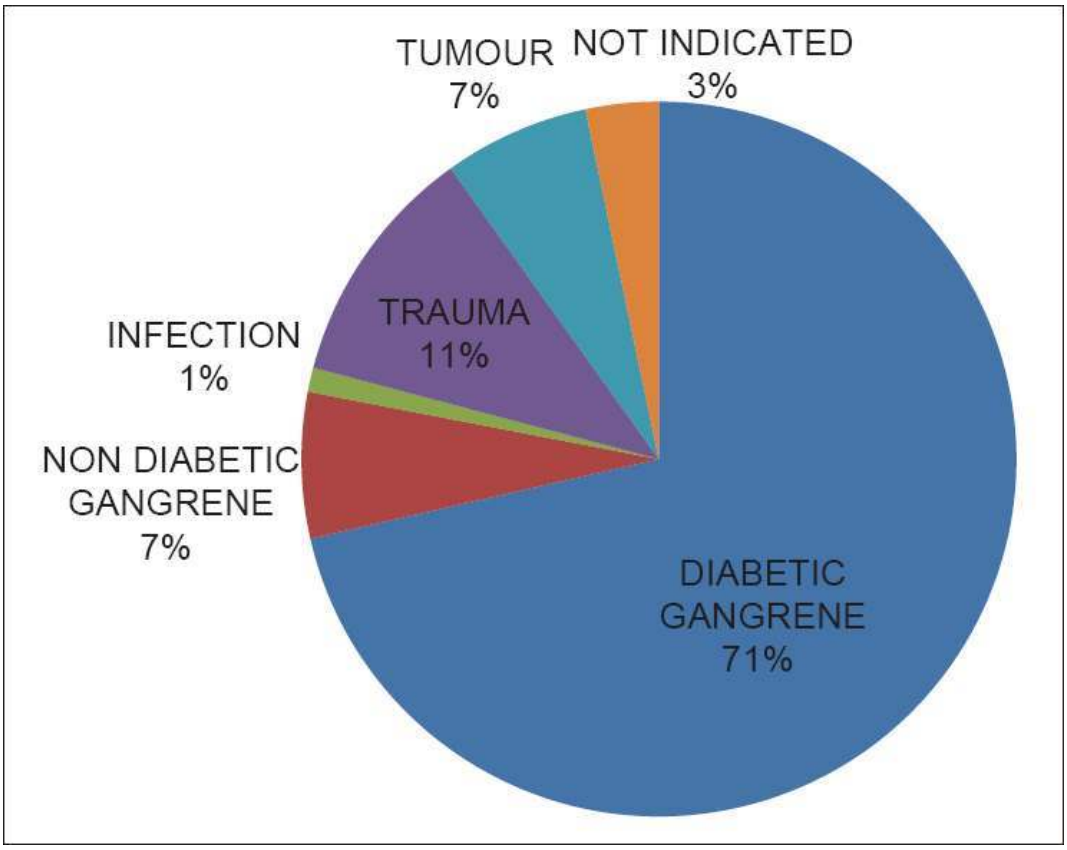
1. Vascular disease

   - Arterial

   - venous

2. Diabetes – 85% of amputation are due to combined diabetes and vascular diseases.
3. Trauma

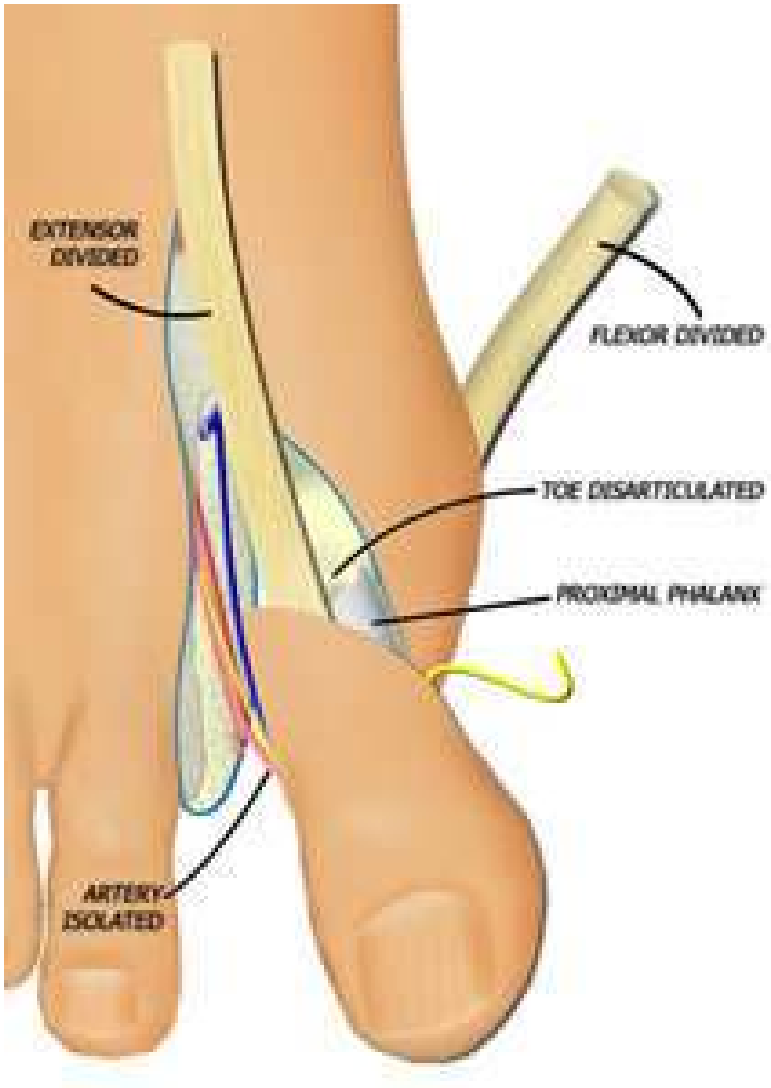
4. Tumours
5. Infection
6. Neurological causes
7. Congenital problem



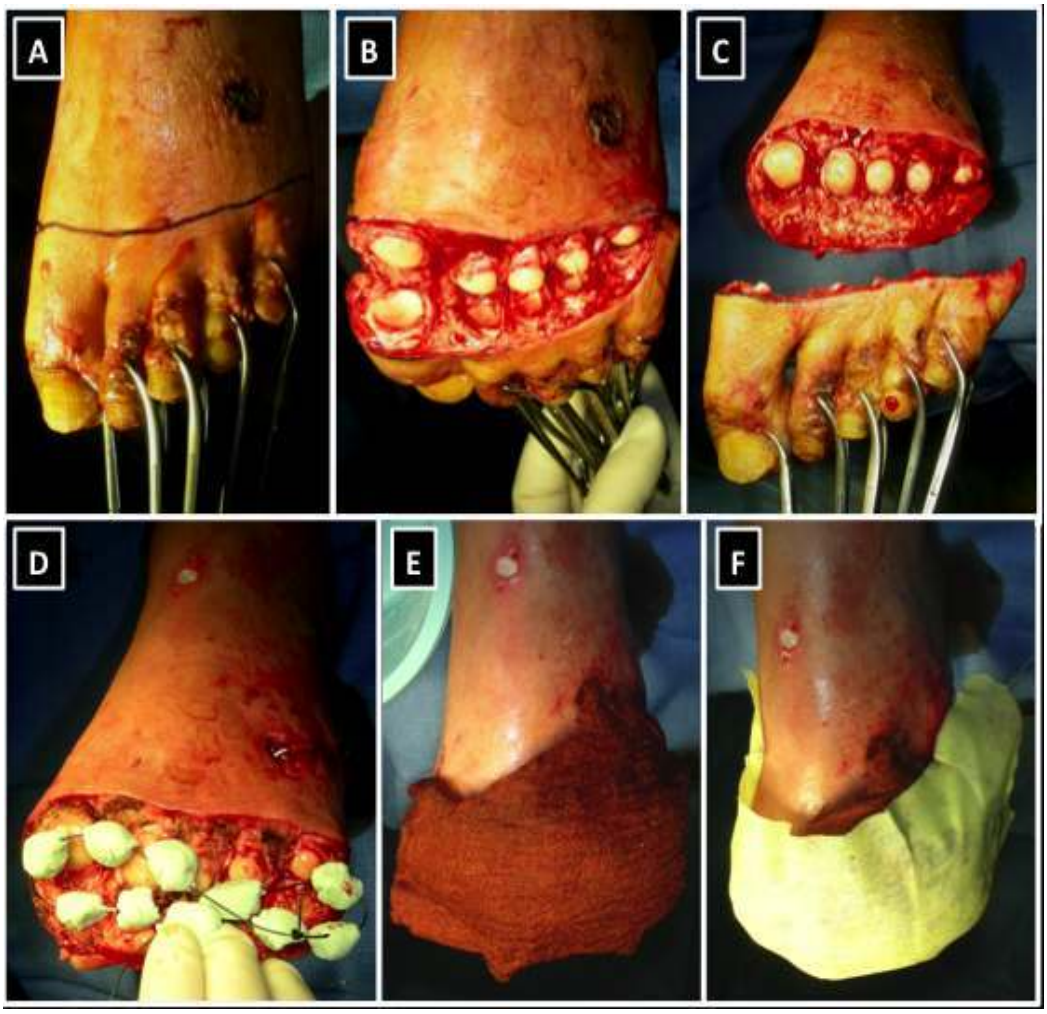
Many risk factors are associated with the chain of conditions and events leading to lower extremity amputation in diabetes. Modification of some of these risk factors by affected subjects and healthcare workers may reduce the risk for amputation and thus decrease the human suffering and monitory loss that follow amputations in very common, chronic disease like diabetes.[26]

Amputations in diabetes can vary from disarticulation of toes, Syme's amputations, ankle level amputations, below knee amputations, above knee amputations and hip disarticulation.

Most common type of amputation in the foot is ray amputation of the affected toe, usually along with the distal half of the corresponding metatarsal. Wound is often left open as this procedure is usually done in infected foot, facilitating drainage.

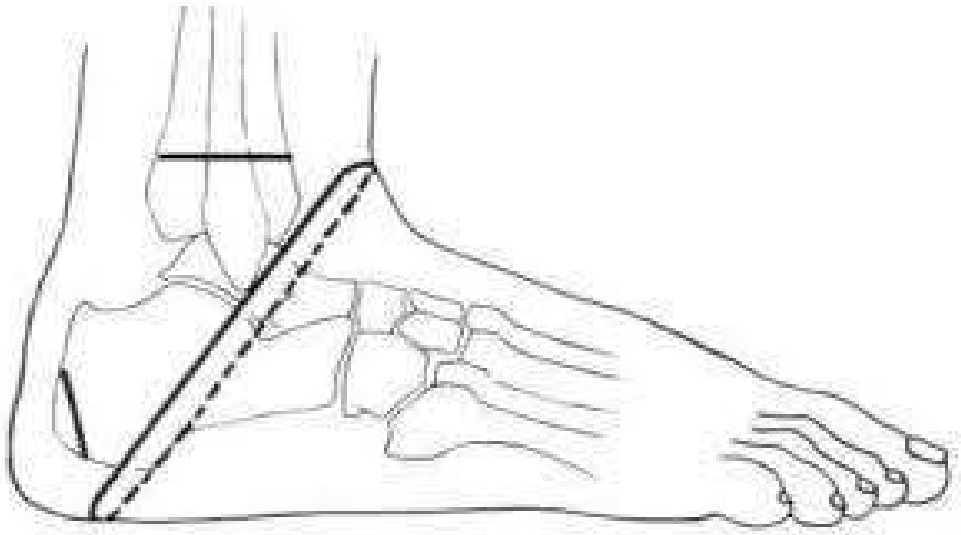


Midtarsal amputations are done for distal gangrenes with adequate hind foot perfusion. In this heel is preserved for weight bearing. A longer plantar flap is used in these cases.

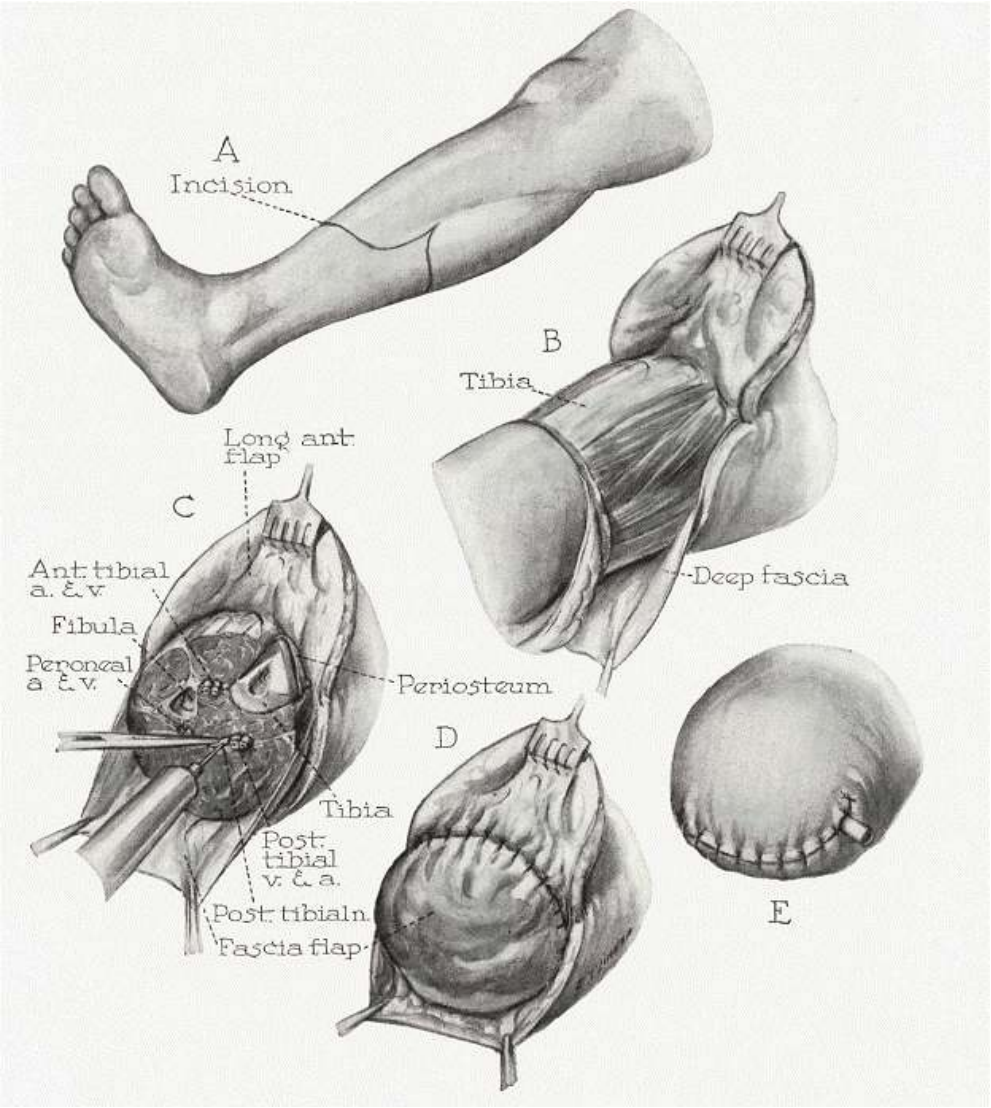


The classical ankle amputation was first described by Syme in 1842. Syme's amputation results in a weight bearing stump and is particularly useful in young individuals. Though the resultant stump is about 5cm short of ground, patient can walk without prosthesis. Modifications of original Syme's procedure, where the distal articular

surface of tibia is left intact and a bigger plantar flap is used, results in better stump length.



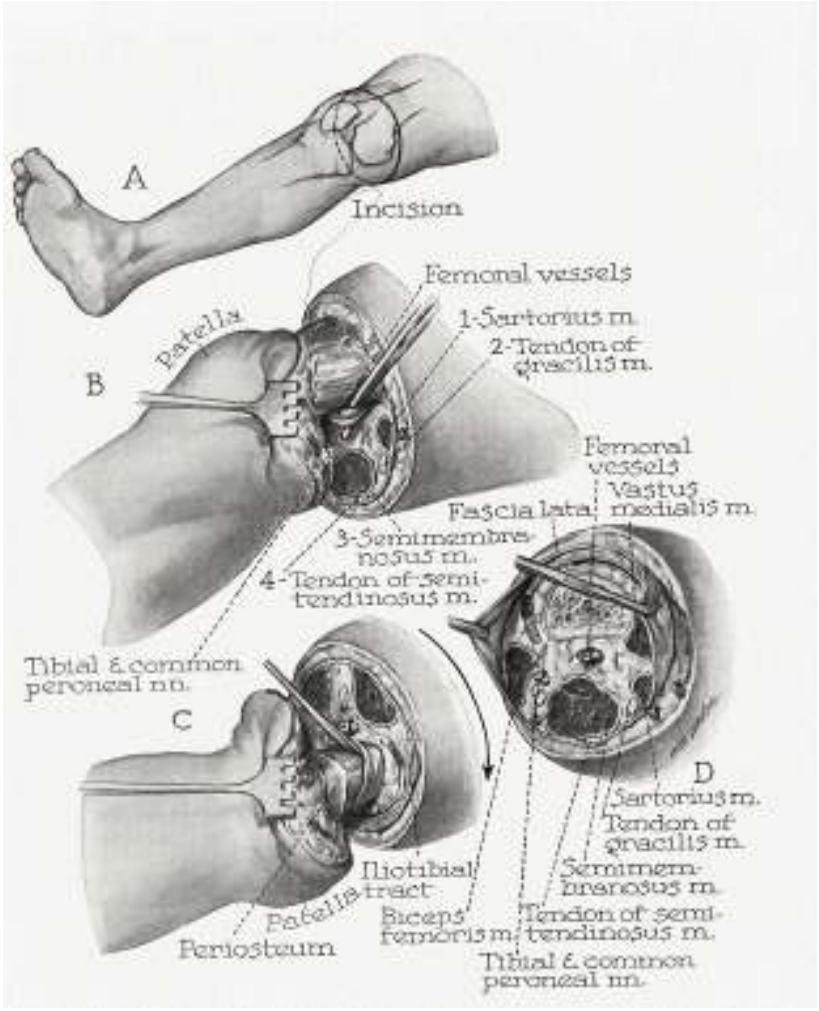
Below knee amputations are the most common procedure done for peripheral arterial diseases. 15cm of tibia is left behind ideally. If the tibial length is less than 8cm, prosthesis fitting may become troublesome. A long posterior flap, 1.5 times the diameter of the leg is used as the anterior flap will have a poorer blood supply. Posterior flap muscles are cut in such a way that only thin layer of gastrocnemius remains.

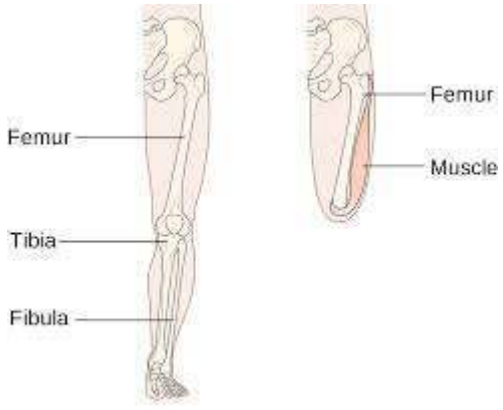


Disarticulation through knee produces a functionally satisfactory, end weight bearing stump. But usually results in a aesthetically unacceptable bulbous end and hence an above knee amputation is usually preferred.

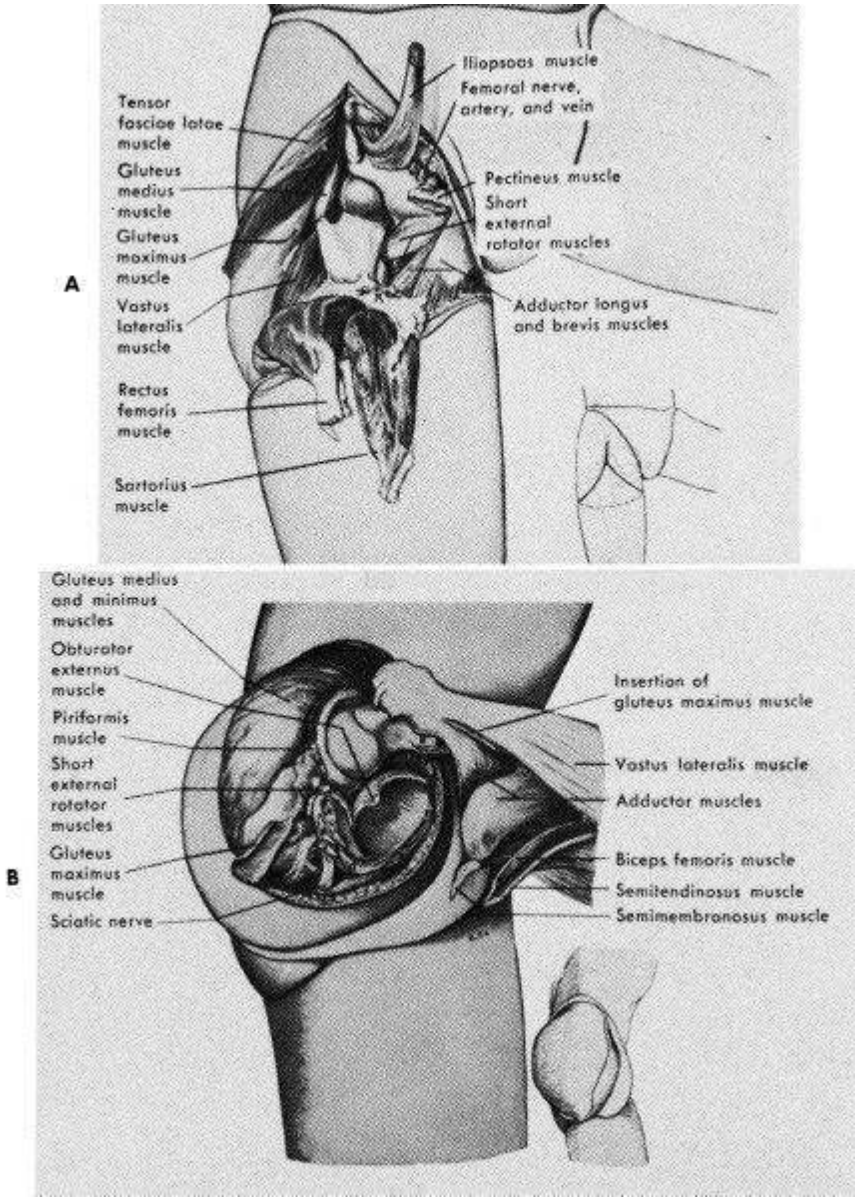
Above knee amputations are common in ischemic peripheral arterial diseases. General dictum is that, longer the stump better is the results. Ideally 70% of the femur, i.e.25-30cm from tip of greater trochanter is retained. Anterior and posterior flaps are equal or a longer anterior flap is used. Hamstrings are sutured to quadriceps for equal muscle action.

Gas gangrene is a dreaded complication especially of above knee amputations for ischaemia.





Radical hip disarticulation is indicated when sufficient length of femur cannot be preserved for a satisfactory prosthesis fixation. Two classical methods are described, anterior racquet methodand a single poster flap method. In both methods femoral vessels are ligated first, muscles are divided and joint exposed and opened. Sciatic nerve is cut short. Capsule is divided and disarticulation is completed.



Hindquarter amputation is a mutilating radical amputation usually done for advanced malignancies. In peripheral vascular diseases it may be done for failed above knee amputations. Fortunately this disfiguring procedure is rarely done.

Complications of amputation

Early complications

1. Haemorrhage
2. Haematoma
3. Inection
4. Gas gangrene
5. Wound dehiscence
6. Gangrene of flaps
7. Deep vein thrombosis
8. Pulmonary embolism

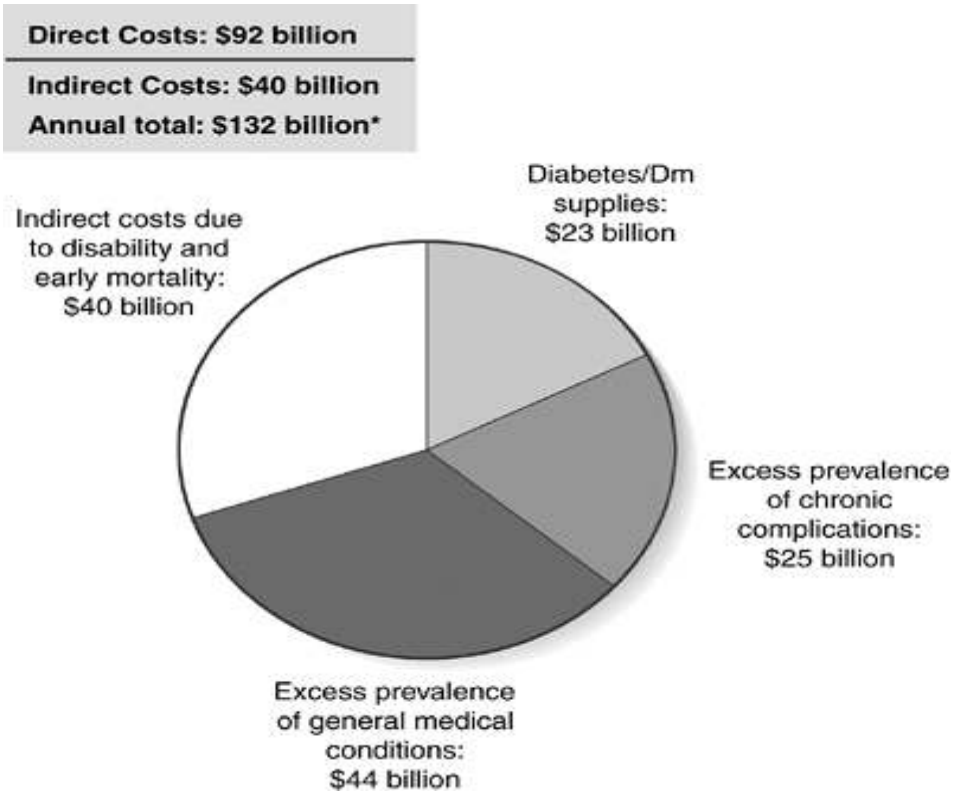
Later complication

1. Unresolved infection
2. Bone spur
3. Scar adherent to bone
4. Amputation neuroma
5. Phantom limb
6. Phantom pain
7. Ulceration of stump

Socioeconomic implications of amputation

Amputations result in physical mental and economic difficulties to the patient and to the society. Sudden loss of mobility or major functions in a previously active individual will result in depression in majority of patients. Loss of productive life results in major economic bereavement both to the individual and to the community.

Post amputation rehabilitation is a very important part of the total diabetic treatment. Indirect losses like loss of productive life, loss in wages, compensations etc will account for about 1/3rd of total diabetic budget of the country.



Prevention of amputations

Prevention of foot ulcers in diabetes can be done by testing for loss of sensation, done easily in the primary care clinic with a short history and the Semmes-Weinstein monofilament. Diabetic speciality clinics may check for neuropathy using biothesiometry, plantar foot pressure measurements, and check lower limd arterial status with hand held Doppler and measurement of ankle-brachial indices. These measurements, along with other clues from the history and clinical evaluation, help practitioners to stratify patients based on risk and to determine the type of intervention. Health education about correct methods of foot care and regular foot examinations are effective methods to prevent diabetic foot. Additional interventions which are effective include optimizing diabetic control, stopping smoking habit, intensive podiatric care, proper care of calluses, proper fitting foot wears avoiding injuries and certain types of prophylactic foot surgery. The value of various types of prescription footwear for ulcer prevention is not clear.[27]

Significant amount of evidence supports screening all patients with diabetes to identify those at risk for diabetic foot. These patients might benefit from certain prophylactic interventions, including patient

education, prescription footwear, intensive podiatric care, and evaluation for any surgical interventions as needed.

Among persons diagnosed as having diabetes mellitus, the lifetime risk of developing a foot ulcer is estimated to be 15%[28].

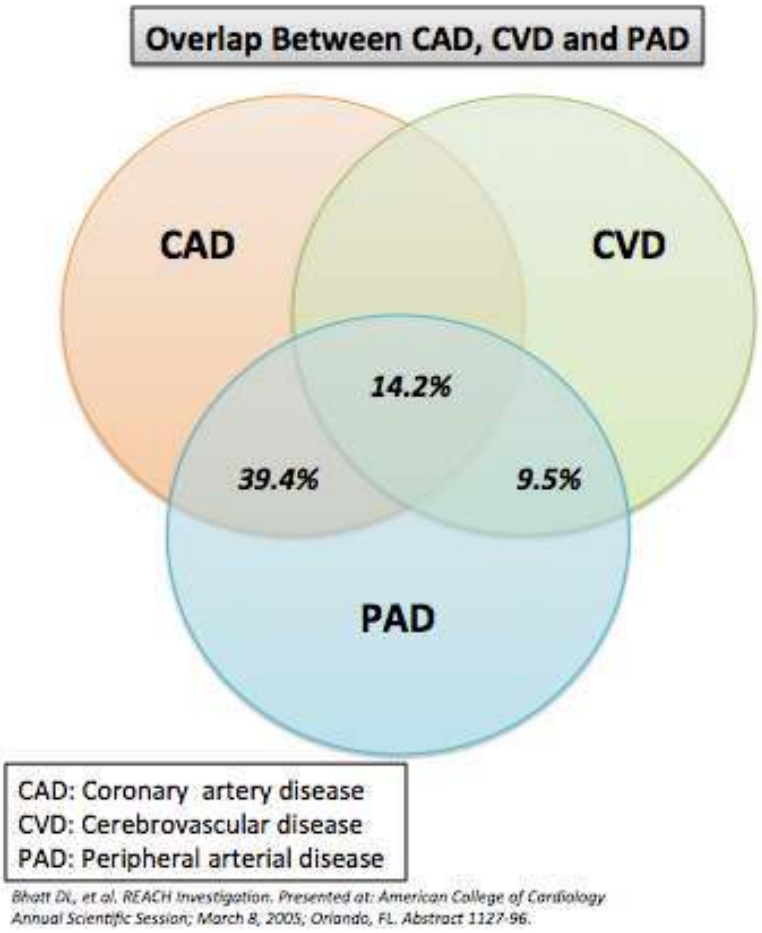
Peripheral arterial disease is most readily detected by the ankle-brachial index (ABI), which is the ratio of systolic blood pressure in the ankle to that in the brachial artery. An ABI of 0.90 or less suggests peripheral vascular disease, while higher than 1.1 may represent a falsely elevated pressure caused by medial arterial calcinosis. This test is easily performed, objective, and reproducible.[29] One large study found that the ABI was strongly related to the risk of foot ulceration (0.3 higher ABI is associated with an RR of 0.83; 95% CI, 0.73-0.96; $P = .01$).[30]

Other vascular diseases and their relation to peripheral arterial disease.

Cardiovascular diseases and strokes are more common in diabetes when compared to non-diabetic individuals. Diabetes mellitus as such, markedly increases the risk of myocardial infarction, stroke, amputation, and death. The metabolic abnormalities caused by diabetes induce vascular dysfunction that predisposes this patient population to atherosclerosis. Control of blood pressure, lipid-lowering treatment,

angiotensin-converting enzyme inhibitors, and antiplatelet drugs significantly reduce the risk of cardiovascular events. Though diabetic patients also undergo revascularization procedures due to acute coronary syndromes or critical limb ischemia, the outcomes are less promising than in nondiabetic people.[31]

Presence of peripheral artery disease, symptomatic or asymptomatic, increases the risk of coronary artery disease and cerebrovascular disease as part of generalized atherosclerosis.



There are several mechanisms by which accelerated atherosclerosis occur in diabetes. Hypertension, abnormal lipids and insulin resistance contribute to the development of atherosclerosis. Hyperglycemia plays a central role in the development of atherosclerosis as evidenced by the occurrence of increased atherosclerosis in persons with poor diabetic control. The overwhelming evidence in literature emphasizes the role of glycemic control as the best possible intervention in the prevention of diabetic complications, both acute and chronic, including peripheral arterial disease.

RESULTS

A total of 100 diabetic patients (cases) and 100 non-diabetic controls participated in the study. All the 100 cases had more than one year type2 diabetes history. Ankle Brachial Index was measured for all 200 participants (both cases and controls). 20 persons (10%) had ABI value <0.9. prevalence off low ABI was significantly higher in diabetics in comparison with non-diabetic controls. (16% vs 4%)

Table-1

Age distribution

	DM N=100	No DM N=100	p-value
Age	51.9 (+/-11.18)	61.37 (+/-9.56)	<0.001

Chart-1

Age distribution

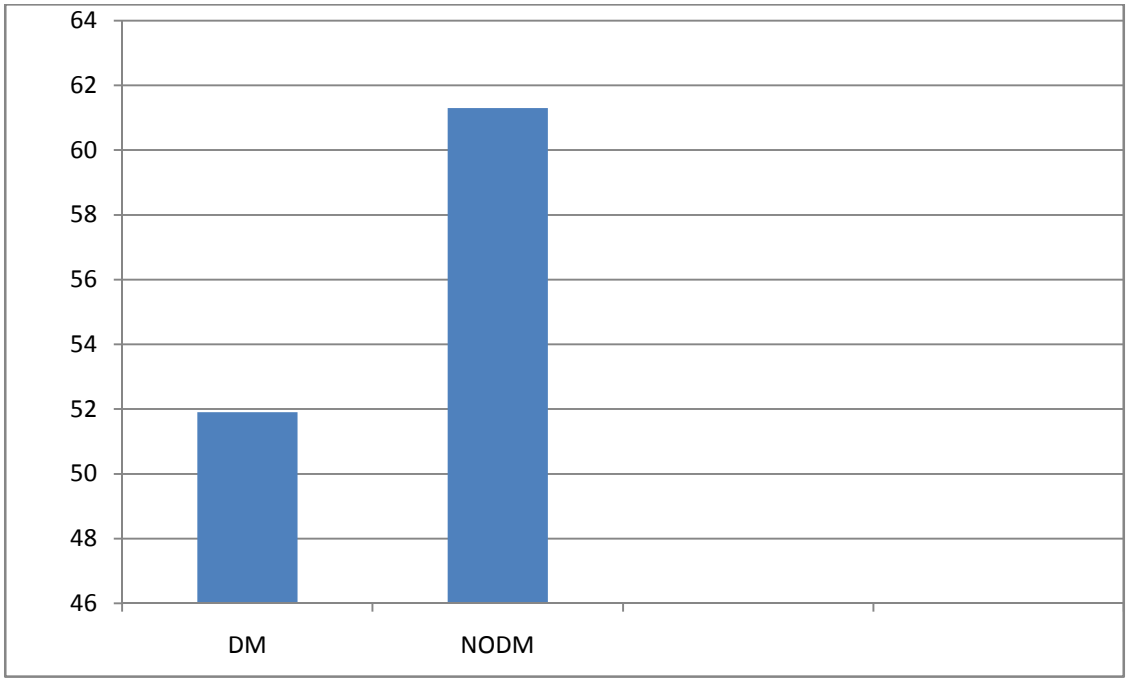


Table 2:

Asymptomatic PAD as evidenced by ABI<0.9

Case/contl	Total N=200	DM N=100	Non DM N=100	p-value
ABI<0.9	20 (10%)	16(16%)	4 (4%)	0.0095

Chart 2:

Asymptomatic PAD as evidenced by ABI<0.9

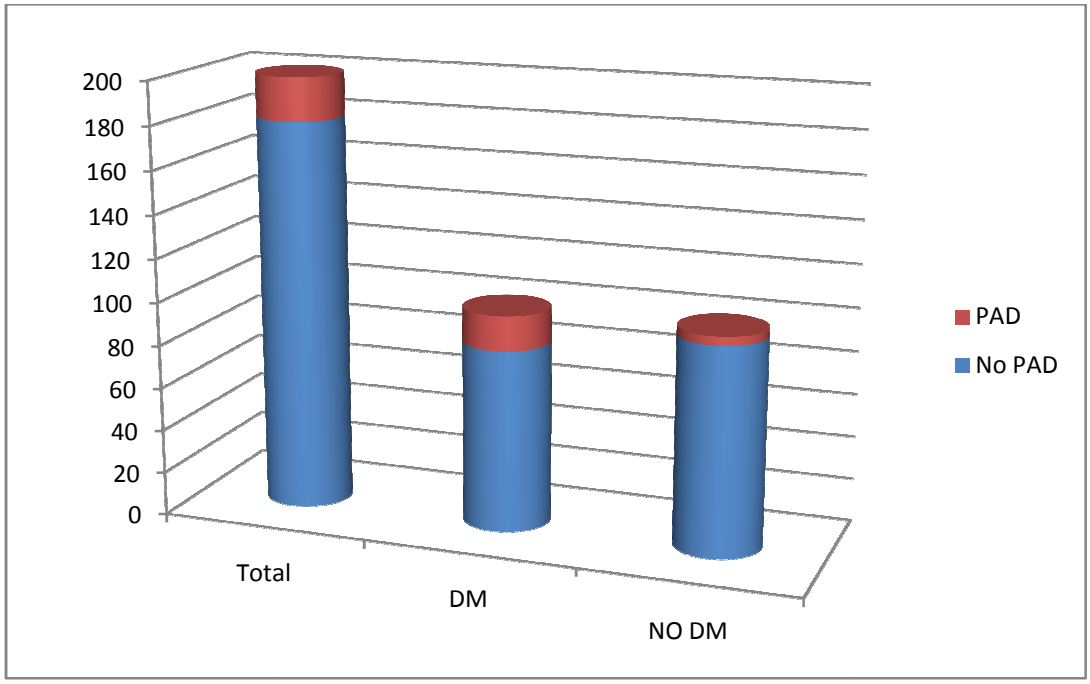


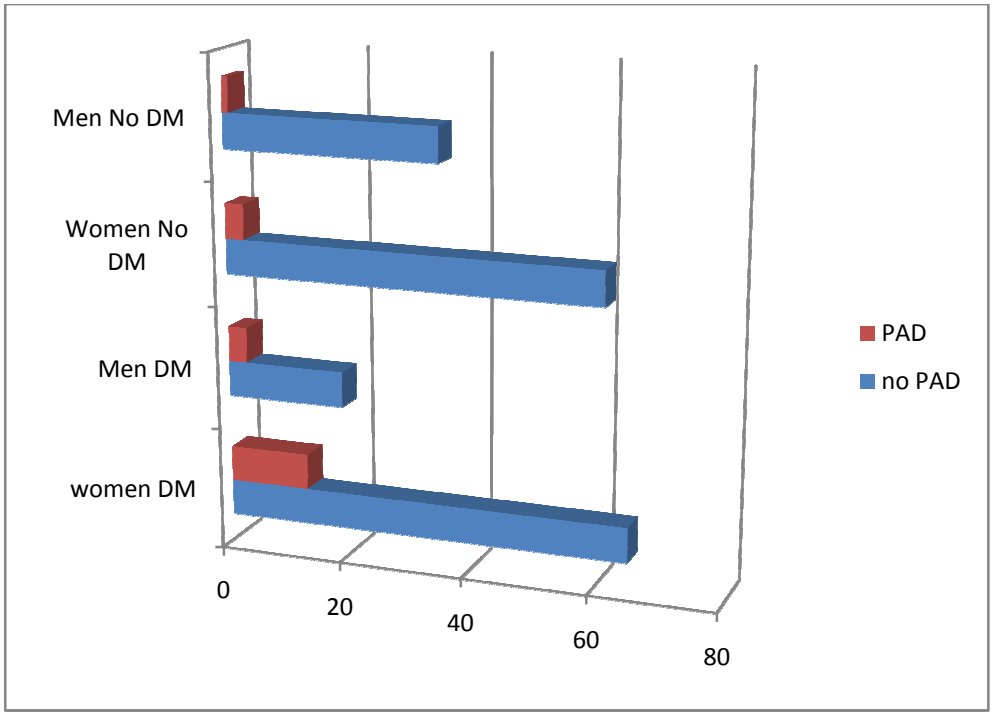
Table 3:

Sex distribution of PAD in Diabetic and Non Diabetic

	Diabetic N=100 (ABI<0.9)	Non DM N=100 (ABI<0.9)	Total N=200 (ABI<0.9)	P
Women	78 (13)	64 (3)	142 (16)	0.0478 (S)
Men	22 (3)	36 (1)	58 (4)	0.2939 (NS)

Chart 3:

Sex distribution of PAD in Diabetic and Non Diabetic



In univariate analysis, age, hypertension, obesity and metabolic syndrome were all associated with low ABI.

Our cohort contained more females than males, probably as a result of the exclusion criteria of tobacco use. The difference between diabetic and non-diabetic women in the prevalence of PAD was statistically significant (p=0.0478), where as the same in men was not statistically significant (p=0.2939). Also, PAD was more common in women both diabetic and non diabetic. This is an important observation when the fact was that a number of these women were passive smokers as their spouses where smoking.

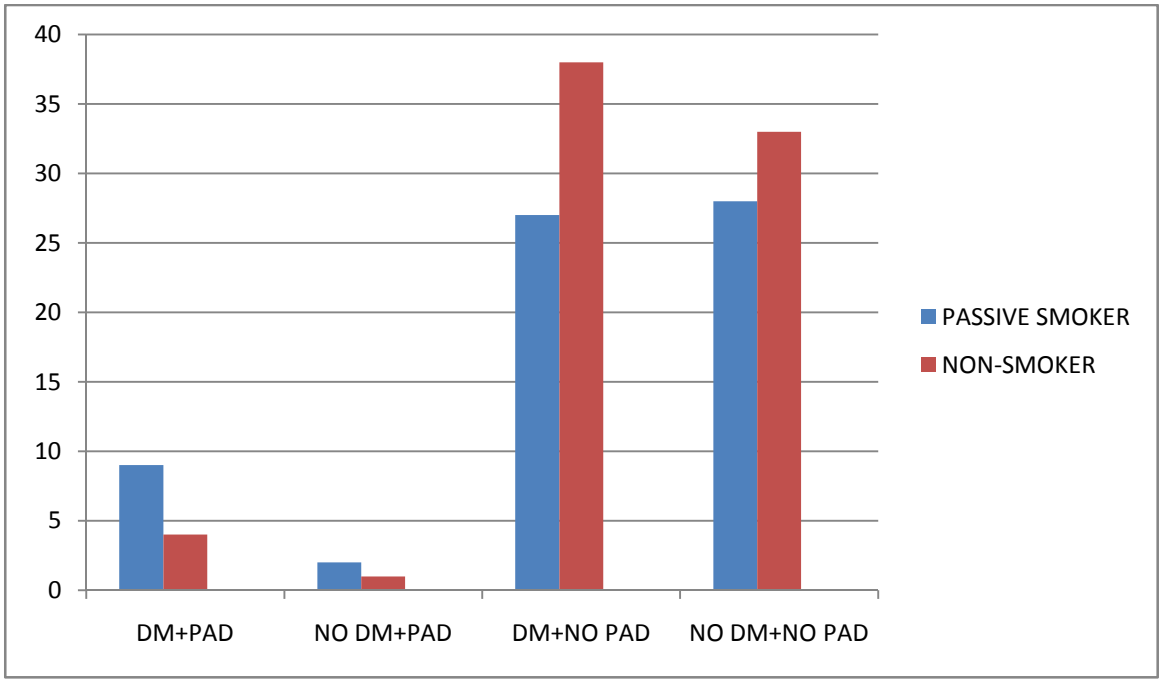
Table 4 :

Relationship of passive smoking with PAD in women

Women	PAD No DM	PAD DM	No PAD DM	No PAD No DM	Total
Passive Smoking	2	9	27	28	66
No smoking	1	4	38	33	76
Total	3	13	65	61	142

Chart 4 :

Relationship of passive smoking with PAD in women



DISCUSSION

According to data collected, there are only a few studies looking into the association of asymptomatic peripheral artery disease to diabetes; though there are a number of studies looking into the association of symptomatic disease with diabetes.

A study by Carlos Lohos etal[32] demonstrated the association between metabolic syndrome asymptomatic peripheral artery diseases, without cardiovascular disease.

In this study all patients with evidence of any significant vascular disease were excluded.

Another study[33] looked into the association of metabolic syndrome and peripheral artery disease in patients already having cardiovascular disease, Peripheral artery disease diagnosis being done by measurement of ankle brachial index <0.9. 14% had low ABI in metabolic syndrome group whereas 10% of those without metabolic syndrome had low ABI.

Yet another study[34] of peripheral artery disease in diabetes reported 12.6% incidence of asymptomatic PAD

A study in South Indian women with type2 diabetes[9], asymptomatic PAD was reported in 19% of diabetic women when compared non-diabetic women of similar age group.

In this study also there were more women included and the incidence of peripheral arterial disease was significantly more in females.

REGICOR investigators demonstrated a 4.5% prevalence of peripheral arterial disease in general population adults[35].

Classical risk factors like age, dyslipidaemia, hypertension are often associated with low ABI[36,37,38]. Many conditions associated with diabetes like low HDL, high Triglycerides, high LDL, metabolic syndrome etc are associated with high incidence of low ankle brachial index and peripheral arterial disease[39].

As per a study by Elizabeth Selvin and Thomas P Erlinger[40], peripheral arterial disease prevalence in adults more than 40 years in the USA was 4.3% (95% CI 3.1% to 5.5%), which translates to ≈5 million persons (95% CI 4 to 7 million). The prevalence was 14.5% (95% CI 10.8% to 18.2%)in elderly, ie those more than 70 years. Black race/ethnicity (OR 2.83, 95% CI 1.48 to 5.42) active smoking (OR 4.46, 95% CI 2.25 to 8.84), diabetes (OR 2.71, 95% CI 1.03 to 7.12), hypertension (OR 1.75, 95% CI 0.97 to 3.13), hypercholesterolemia (OR

1.68, 95% CI 1.09 to 2.57), and low kidney function (OR 2.00, 95% CI 1.08 to 3.70) were positively associated with prevalent PAD in age and sex adjusted multivariate analysis.

Gender difference was variable in published literature. In a review by Higgins and Higgins,[41] in women 45 to 93 years of age had a 3% to 29% (over this span of 5 decades) prevalence of peripheral arterial disease. But, it was evident that peripheral arterial disease was common in diabetic women. Most of these studies were done with cohort including smokers also. More men than women were smokers and hence, naturally men had higher incidence of peripheral arterial disease. We excluded all smokers, both men and women and found that women had higher incidence of peripheral arterial disease in both diabetic and nondiabetic populations, which was probably related to passive smoking as shown by the analysis.

Allison et al[42], showed that the prevalence of peripheral arterial disease increased with age for both men and women. More than just definitions, any atherosclerotic disease had higher occurrence, i.e., increase in the population "burden" of these diseases (defined as the total number of individuals who have the disease).

Age, the most traditional risk factor for peripheral arterial disease was seen to increase incidence[43]. However in our study, diabetic population was younger than control population ($p<0.001$).

ABI is useful as a marker for atherosclerotic risk factors and also vascular diseases in other vascular beds. Low ankle-brachial index is associated with a number of other risk factors, like hypertension, type2 diabetes, dyslipidemia, history of smoking, and several other cardiovascular risk factors (e.g., high sensitivity CRP, interleukin-6, homocysteine, and chronic kidney disease)[44].

Death and the composite end points of stroke or myocardial infarction (MI) occurred in 8.4% and 11.6% of patients. Bad prognosis was noted in patients with prior history of CHD, extremes of age, people with diabetes and a low ankle brachial index as shown by a report from United Kingdom by Gerard Stansby, MChir et al[45].

As a marker of future cardiovascular events, stroke events and amputations; asymptomatic peripheral arterial disease is an important, simple clinical tool, though terribly underutilized. More and more studies in the field will overemphasize the utility of ankle-brachial index as a regular screening procedure in diabetics with high sensitivity and specificity preventing future morbidity and mortality.

Higher incidence of peripheral arterial disease in diabetes was demonstrated previously in a number of studies and is a well known factor. Higher incidence of asymptomatic peripheral artery disease demonstrated in our study is in concordance with available previous studies.

SUMMARY

Diabetes mellitus is a very common disease all over the world and especially so in India. Peripheral arterial disease is a very common macro vascular complication of diabetes and can result in morbidities like chronic ulcers, amputation etc.

Ankle-brachial index, the ratio of ankle pressure to the brachial pressure is an easily done clinical test with sufficient sensitivity and specificity and is easily reproducible.

Only simple tools like sphygmo manometer and a doppler probe are required for doing this test. A low ankle brachial index diagnoses asymptomatic peripheral arterial diseases in patient who are otherwise asymptomatic.

100 patients with diabetes and 100 controls without diabetes were included in this study. All those with pre existing peripheral arterial diseases, leg ulcers, claudication, chronic leg pain syndrome coronary artery disease and stroke were excluded. All tobacco users were also excluded.

- Age wise diabetic patients were younger (Mean 51.9 $\pm$ 11.18 years) than non diabetics (Mean 61.37 $\pm$ 9.56 years).

- 16 (n = 100) diabetic patients and 4 (n = 100) non diabetic patients had ABI < 0.9 (P = 0.0095).
- Both the cohorts consisted mostly of females (all tobacco users were excluded) females had higher incidence of peripheral arterial diseases (female 13/78 vs 3/64 P = 0.0478) (males 3/22 vs 1/36 P =0.2939)

Passive smoking assessed by the presence of active smoker in family was seen in 66 out of 142 women in the study ((36 diabetics and 30 non diabetics)and peripheral arterial disease was common in these passive smoker both in diabetics (9/36) non diabetics 2/30)

Inference of this study is that asymptomatic peripheral artery diseases is common in diabetics and among diabetics more common in females. Passive smoking is an important risk factors for females with peripheral artery diseases.

CONCLUSIONS

1. Peripheral artery disease is common in diabetes, both men and women.

2. The condition may not be detected in many patients due to absence of signs and symptoms even when the blood flow limitation is significant.

3. Though asymptomatic, these patients with low ABI are potentially at risk of developing ischemic limb and associated complications.

4. Other vascular complications like coronary artery disease and cerebrovascular disease are also fairly common in these patients and hence detection of asymptomatic disease is important to prevent complications.

5. Females were more included in the study as a result of the exclusion criteria, smoking, which excluded most men. However, among those included, men had a lower incidence of peripheral arterial disease compared to females. Analysis of history revealed that most of these females had a smoking partner at home, making these ladies passive smokers.

BIBLIOGRAPHY

1. Frowkes FG.The measurement of atherosclerotic peripheral arterial disease in epidemiological surveys.Int J Epidemiol 17:248-254 1988

2. Dachun Xu, Jeu Li, Liling Zou, Ya wei Zu, Dayi Hu, Pagoto SL, Yunsheng Ma].Sensitivity and Specificity of ABI to diagnose peripheral arterial disease: a structural review. Vasc Med 2010 oct; 15(5):361-9

3. cheng sw,Ting AC, Lau H,Wong J): Survival in patients with chronic lower extremity ischemia:A risk factor ananlysis.Ann vasc surgery 14:158-165,2000

4. Pyorala K,laakso M, Unsitupa M: Diabetes and atherosclerosis:an epidemiological view. Diabetic Metab Rev 3:463-524,1987. Donahue RP,Orchard TJ: Diabetes mellitus and macrovascular complications: an epidemiological perspective.Diabetes care 15:1141-1155,1992

5. Chapter 30: Type 2 Diabetes Mellitus; John B. Buse, Kenneth S.Plonsky, and Charles F. Burant. Williams Textbook of Endocrinology; 11th Edition. Editors: Henry M. Kronenberg, Sholomo Melmed, Kenneth S.Plonsky, P. Reed Larsen. Saunders Elsevier 2008

6. chapter 344, Diabetes mellitus,Alvin C Powers; Harrisons Principle of Internal Medicine 18th edition: Dan L Longo, Anthony S Fauci, Dennis L Kasper, Stephen L Hausen , J larry Jameson, Joseph Loscalzo. Mc Graw Hill medical

7. Mohan V, Sandeep S, Deepa R, Shahand Varghese. Epidemiology of Type2 Diabetes; Indian scenario. The Indian Journal of Medical Research 2007; 125(3) pp217-30. ISSN-0971-5916

8. King H,Aubert R, Heman W. Global burden of Diabetes 1995-2025, Prevalence, Numerical estimates and Projections. Diabetes Care 1998; 21: 1414- 1431

9. M G Binu, P Shanija, J Bino John Sahayo. Asymptomatic Peripheral Artery Disease in South-Indian Women with Type2 Diabetes; Indian J Endocrinol Metab Jul 2011; 15 (suppl 1): S68 - 69

10 Chapter-23, Arterial Disease pp704-705, Schwartz's Principles of Surgery, 9th edition, 2010. Editors: F Charles Brunicardi, Dana K. Andersen, Timothy R. Billiar, David L. Dunn, John G. Hunter, Jeffrey B. Matthews,Raphael E. Pollock. Mc Graw Hill Medical

11 Carlos Lahos MD etal.Metabolic syndrome and asymptomatic peripheral artery disease in subjects over 60 years of age.Diabetes care 29:148-150 2006

12 Chapter 12, Pathogenesis of Diabetic Complications pp168-187. A Handbook of Diabetes Mellitus, Third edition. 2007. Editor: V Seshaiah. All India Publications

13 Chapter 32; Complications of Diabetes Mellitus; Michael Brownlee, Lloyd P.Aiello, Mark E.Cooper, Aaron I.Vinik, Richard W.Nesto, and Andrew J.M.Boulton. Williams Textbook of Endocrinology; 11th Edition. Editors: Henry M. Kronenberg, Sholomo Melmed, Kenneth S.Plonsky, P. Reed Larsen. Saunders Elsevier 2008.

14 Chapter 10, Lipids and Diabetes Mellitus. Handbook on Diabetes; Editor: Prof. V.Sesiah, Third edition pp:145-158. 2007 All India Publishers.

15 Rury R. Holman, Sanjoy K Paul, Angelin Bethel, David R Mathews and Andrew W Neil. 10-year follow of intensive glucose control in Type 2 diabetes N Engl J Med 2008 ; 359:1577-1589/October 9, 2008 / DOI : 10.105/NEJM0a0806470.

16 M.G.Binu et al. Role of lipid profile in the short term prognosis of acute myocardial infarction in a rural hospital in South India Appolo Medicine 2012 September, vol 9, Number 3, pp:263-267

17 Kannel WB, McGee DL. Diabetes and glucose tolerance as risk factors for cardiovascular disease: the Framingham study. Diabetes care 1979;2:120-126

18 Brand FN,Abbot RD,Kennel WB. Diabetes, intermittent claudication and risk of cardiovascular event. The Framingham study. Diabetes 1989;38:504 -509

19 Kreines K, Johnson E, Elbrink M,et al.the course of peripheral vascular disease in non-insulin dependent diabetes.Diabetes care 1985;8:235-243

20 Melton LJ,Machen KM,Palumba PJ Eleveback LR . Incidence and prevalence of clinical peripheral vascular disease in a population based cohort of diabetic patients. Diabetes care 1980;13:650-654

21 Nilsson SV,Nilsson JE,Frostberg N,Emilsson T. the Kristianstand study II. Acta Med Scand 1967;469: 1-42

22 Janka HV,Standl E,Mehnet H.peripheral vascular disease in diabetes mellitus and its relation to cardiovascular risk factors. Screening with the Doppler ultrasonic technique. Diabetes care 1980;3 207-213

23 Neil HAW,Thompson AV,Thorogood M,et al.Diabetes in the elderly:the Oxford community diabetes study. Diabet Med 1989;6:608-613

24 Orchard TJ,Dorman JS,Maser RE,et al.prevalence of complications in IDDM by sex and duration.Pittsburg Epidemiology of Diabetes complications study II. Diabetes 1990;39:1116-1124

25 Coce F,Jaksic B, Jaksic A.Faktori rizika dijabeticke obliterative angiopatije.Diabetol Croat. 1982; 2:337-340

26 Gayle E. Reiber, MPH, PhD; Roger E. Pecoraro, MD; and Thomas D. Koepsell, MD, MPH. Risk Factors for Amputation in Patients with Diabetes Mellitus: A Case-Control Study; *Ann Intern Med.* 1992;117(2):97-105. doi:10.7326/0003-4819-117-2-97

27 Preventing Foot Ulcers in Patients With Diabetes. Nalini Singh, MD; David G. Armstrong, DPM, MSc, PhD; Benjamin A. Lipsky, MD *JAMA.* 2005;293(2):217-228. doi:10.1001/jama.293.2.217

28 Reiber GE. The epidemiology of diabetic foot problems. *Diabet Med*. 1996;13:(suppl 1) S6-S11

29 American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care*. 2003;26:3333-3341

30 Boyko EJ, Ahroni JH, Stensel V, Forsberg RC, Davignon DR, Smith DG. A prospective study of risk factors for diabetic foot ulcer: the Seattle Diabetic Foot Study. *Diabetes Care*. 1999;22:1036-1042

31 Diabetes and AtherosclerosisEpidemiology, Pathophysiology, and Management Joshua A. Beckman, MD, MS; Mark A. Creager, MD; Peter Libby, MD *JAMA*. 2002;287(19):2570-2581. doi:10.1001/jama.287.19.2570.

32 Carlos Lohos.MD etal. Metabolic syndrome and asymptomatic peripheral artery disease in subjects over 60 years age. Diabetes Care 29: 148-150; 2006

33 Olighoek JK et althe metabolic syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease and abdominal aortic aneurysm. Eur Heart J 25:342-348; 2004

34 Franjo Coce etal. Peripheral arterial diasease and diabetis mellitus. Diabetologia Croatica, 37-2: 47-53; 2008

35 R Ramos, M Quesada etal on behalf of the REGICOR investigators, Prevalence of symptomatic and asymptomatic peripheral arterial disease and the value of ankle brachial index to stratify cardiovascular risk, European Journal of Vascular and Endovascular Surgery. September 2008, vol38(3): 305-311

36 Peripheral arterial disease in people with diabetes. Consensus statement. American Diabetes Association. Diabetes Care 26: 3333-3341 2003

37 Mourabito J M et al. Prevalence and clinical correlates of peripheral arterial disease in the Framingham offspring study. Am Heart J 143: 961-965; 2002

38 Curb J D et al. Peripheral artery disease and cardiovascular risk factors in elderly: Honolulu Heart Program. Arterioscler Thromg Vasc Biol 16: 1495-1500; 1996

39 Jurenne D Hooi, Henri EJH Stoffers, Arnold DM Knotteneras. Risk factors and cardiovascular disease associated with asymptomatic peripheral arterial occlusive disease: The Limburg PAOD study. Scandinavian Journal of Primary Health Care 1998, vol 16, issue 3: pages 177-182

40 Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in united states. Circulation 2004; 110: 738-743.

41 Higgins JP, Higgins JA. Epidemiology of peripheral arterial disease in women. J Epidemiol. 2003;13:1–14.

42 Allison MA, Ho E, Denenberg JO, Langer R, Newman AB, Fabsitz RR, CriquiMH. Ethnic-specific prevalence of peripheral arterial disease in the United States.Am J Prev Med. 2007;32:328–333.

43 Anderson JD, Epstein FH, Meyer CH, Hagspiel KD, Wang H, Berr SS, HarthunNL, Weltman A, Dimaria JM, West AM, Kramer CM. Multifactorial determinants of functional capacity in peripheral arterial disease: uncoupling of calf muscle perfusion and metabolism. J Am Coll Cardiol. 2009;54:628–635.

44 Gerard Stansby, MChir FRCS High Risk of Peripheral Arterial Disease in the United Kingdom: 2-Year Results of a Prospective RegistryANGIOLOGY February 2011 vol. 62 no. 2111-118

APPENDIX

PROFORMA

INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL

1. Name of the Patient :
2. Age :
3. Sex :
4. Address :
5. Income :
6. Diabetes : Yes / No

   If yes ……. year
7. History of HT / CAD / CVA / POVD / Diabetic Ulcer / Dyslipidaemia / Renal Failure
8. Treatment History : Atorvastatin / Betablocker
9. Smoking : Actve / Passive / None
10. Other Tobacco use
11. Alcohol Use
12. Examination Anaemia Oedema
13. Pulse Rate Peripheral Pulses Vessel Wall
14. CVS Examination
15. Nervous System Peripheral Neuropathy – Position Sense

Vibration Sense Monofilament

16. Abdomen & Respiratory System
17. Blood Pressure (Mannometer & Stethoscope)
18. Brachial Pressure with Doppler
19. Ankle pressure with Doppler
20. Ankle brachial index
21. Egg
22. Lipid Profile
23. FBS PPBS UREA CREATININE

CONSENT FORM

I am **Dr. VINODINI .C**, carrying out a study on the topic **"INCIDENCE OF ASYMPTOMATIC PERIPHERAL ARTERY DISEASE IN TYPE 2 DIABETES IN A TERTIARY CARE HOSPITAL"** in Government Medical College and Hospital, Coimbatore conducted by Dr. C. Vinodini, Post Graduate Student in General Surgery Department, Coimbatore Medical College you satisfy eligibility criteria for inclusion. You can feel free to ask any question or seek any further clarification on the study and clear any doubts that you may have, before agreeing to participate.

My research project is being carried out under the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

RESEARCH BEING DONE:

To determine the Incidence of asymptomatic Peripheral Artery Disease in Type 2 Diabetics with the help of BP apparatus and hand-held Doppler, calculating the Ankle Bracheal Index.

PURPOSE OF THE RESEARCH

To find out the incidence of asymptomatic peripheral artery disease in type 2 diabetic individuals and compare it with general population.

PROCEDURE INVOLVED

Ordinary digital sphygmomanometer and a hand-held Doppler device are used to measure brachial and ankle blood pressure. Diabetic control is assessed as per the blood sugar reports. No invasive tests or treatments will be undertaken as part of study.

DECLINING FROM PARTICIPATING

You are hereby made aware that, participation in this study is purely voluntary and honorary and that you have the option and right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about the privacy. Privacu of the subject will be respected and any information about you or provided by you durin study will be kept strictly confidential.

AUTHORISATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy breached

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by **Dr. C. VINODINI**. I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date:

Place:

Signature and Name of witness Date:

Place:

Signature of the investigator:

Name of the investigator:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் :

முகவரி : வயது :

அரசு கோவை மருத்துவக் கல்லூரியில், பொது அறுவை சிகிச்சை துறையில், பட்ட மேற்படிப்பு பயிலும் மாணவி மரு. வினோதினி அவர்கள் மேற்கொள்ளும் "சர்க்கரை நோயாளிகளில் எத்தனை பேருக்கு அறிகுறியில்லாத இரத்த நாள நோய் உள்ளது" என்ற சோதனையின் செய்முறை மற்றும் அனைத்து விபரங்களையும் கேட்டுக்கொண்டதுடன், எனது அனைத்து சந்தேகங்களையும் தெளிவுப்படுத்திக்கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன்.

நான் இந்த ஆய்வில் முழு சம்மதத்துடனும், சுய சிந்தனையுடனும் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விபரங்களும் பாதுகாக்கப்படுவதுடன், இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் எனக்கு எந்த ஆட்சேபனையும் இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து விலகிக்கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

இடம் :

தேதி : **கையொப்பம் / ரேகை**

MASTER CHART

CASES

S.No	Name	Age	Sex	ABI	PS/NS
1	Krishnaveni	42	F	1	PS
2	Malathi	46	F	1.2	NS
3	Suganthi	62	F	1.156	NS
4	Krishnan	38	M	1.3	NS
5	Gopi	54	M	0.86	NS
6	Kanchana	51	F	0.82	PS
7	Esrael	56	M	1.16	PS
8	Gokila	50	F	1.07	NS
9	Sankar	72	M	0.9	NS
10	Sowdambika	44	F	1.15	NS
11	Revathy	69	F	0.842	PS
12	Sankari	48	F	1.33	NS
13	Latha	67	F	0.85	NS
14	Veni	75	F	1.07	PS
15	Sarawathi	42	F	1.36	NS
16	Lakshmanan	56	M	1.45	NS
17	Getha	55	F	0.9	PS
18	Krishnagounder	70	M	0.94	NS
19	Sudha	43	F	0.85	NS
20	Ramasamy	60	M	1.43	NS
21	Kittan	63	M	0.93	NS
22	Jaisankar	57	M	1.89	NS
23	Tamilseli	70	F	1.25	NS
24	Parvathi	35	F	0.87	PS
25	Sreenivasan	65	M	0.97	NS

26	Durga	68	F	0.57	PS
27	Ruckmani	57	F	1.07	PS
28	Poovathal	47	F	0.88	NS
29	Elango	50	M	0.96	NS
30	Lakshmi	38	F	1	PS
31	Santhi	41	F	1	PS
32	Kathirvel	50	M	1.07	NS
33	Chinnasamy	42	M	1.33	NS
34	Usha	53	F	1.45	NS
35	Nanthini	50	F	0.93	PS
36	Thulasi	40	F	1.07	NS
37	Pappammal	49	F	1.2	NS
38	Nagaraj	60	M	1.07	PS
39	Ramathal	49	F	1.08	PS
40	Pankajam	58	F	1.12	NS
41	sivajothi	40	F	1.06	NS
42	Ramakkal	42	F	0.92	PS
43	Renuga	60	F	0.95	NS
44	Balu	75	M	1.35	NS
45	Banumathy	48	F	1.16	NS
46	Devaki	42	F	1.19	NS
47	Krishnammal	75	F	1.86	NS
48	Arumugam	53	M	1	PS
49	Devi	66	F	0.79	NS
50	Papai	57	F	1.29	NS
51	Swaminathan	51	M	1.36	NS
52	Thilaga	38	F	1	PS
53	Hyanemicha	41	F	0.82	PS

54	grachy	60	F	0.9	PS
55	Sarasakka	57	F	1	NS
56	Backiyam	65	F	1.29	NS
57	Kumari	62	F	1.16	PS
58	Murugesh	68	M	1.2	NS
59	Ambika	62	F	1.25	PS
60	Jaya	54	F	1.125	PS
61	Govindan	45	M	1.214	NS
62	Manickam	60	M	1.57	NS
63	Navaneetham	54	F	0.88	NS
64	Chellakkal	64	F	1.5	NS
65	Sarawathy	57	F	1.09	PS
66	Indira	67	F	0.8	PS
67	Selvaraj	50	M	0.8	NS
68	Banumathy	54	F	0.93	NS
69	Shantha	60	F	1.067	NS
70	Jagathambal	63	F	1	PS
71	Mrijeebunisha	66	F	1.2	PS
72	Kaliyamma	55	F	1.412	NS
73	Ganga	59	F	1.15	NS
74	Gopinath	33	M	1.2	NS
75	Savithri	42	F	0.96	PS
76	Gomathi	43	F	0.93	PS
77	Karthika	35	F	1.89	NS
78	Malini	39	F	1.25	NS
79	Subbammal	62	F	0.76	PS
80	Revathi	32	F	0.94	PS
81	Thulasi	44	F	1.45	NS

82	Valli	32	F	1.36	NS
83	Lathadevi	38	F	1.45	NS
84	Mariamma	56	F	0.86	NS
85	Devipriya	46	F	0.93	NS
86	Hamsathbegam	58	F	0.85	NS
87	Julie	50	F	0.95	NS
88	Mary	43	F	1	PS
89	Lakshmi	34	F	1.07	PS
90	Kanagam	48	F	1.33	NS
91	Gowri	41	F	0.97	NS
92	Krishnakumari	64	F	0.78	PS
93	Bakiyalakshmi	34	F	1.29	NS
94	Gomathi	43	F	1.16	NS
95	Parithunisha	55	F	0.8	PS
96	Rohini	35	F	1	NS
97	Kaliyammal	50	F	0.94	PS
98	Fathimabeevi	43	F	1.066	PS
99	Sivasankari	48	F	1.15	NS
100	Sowmya	36	F	0.96	PS

CONTROL

S.No	Name	Age	Sex	ABI	PS/NS
1	Kannan	74	M	1.25	NS
2	Sugantha	60	F	1.43	NS
3	Srikumari	50	F	0.9	PS
4	Jaya	42	F	1.36	NS
5	Gopalsamy	68	M	0.94	NS
6	Janaki	63	F	0.93	PS
7	Ganga	59	F	1.2	NS
8	Ismail	65	M	1.1	NS
9	Marathal	72	F	0.98	PS
10	Mohan	44	M	1.21	NS
11	Mariammal	65	F	1.45	PS
12	Ramachandran	53	M	1	NS
13	Palana	41	F	0.99	PS
14	Pappathi	75	F	0.96	PS
15	Sivasankari	66	F	1.2	NS
16	Kuttiakka	58	F	1.14	NS
17	Ramkumar	54	M	1.18	NS
18	Pankajamma	72	F	1.22	NS
19	Paramasivam	43	M	0.99	NS
20	Periyakka	75	F	0.92	PS
21	Kunthi	59	F	1.3	NS
22	Rosammal	68	F	1.21	PS
23	Subbammal	62	F	1.2	PS
24	Rasatbi	66	F	1.14	NS
25	Kanagam	71	F	1.08	PS
26	Ganesh	40	M	1.2	NS

27	Rammathal	63	F	0.85	PS
28	Lakshmiammal	67	F	1.02	NS
29	Parvathy	54	F	1.18	NS
30	Tamilselvi	49	F	0.92	PS
31	Kannan	68	M	0.96	PS
32	Gracyamma	70	F	1.15	NS
33	Padmaraj	66	M	1.14	NS
34	Kuppammal	69	F	1.26	PS
35	Karthikeyan	73	M	1.28	NS
36	Premalatha	59	F	1.06	PS
37	Venkatesh	64	M	1.08	PS
38	Rajesh	61	M	1.36	NS
39	Joe	63	M	1.07	NS
40	Maheshwari	39	F	0.94	PS
41	Bhavani	74	F	0.98	NS
42	Ramanathan	65	M	0.99	NS
43	Sasikala	69	F	1.36	NS
44	Janaki	60	F	0.94	PS
45	Sakthi	58	F	0.98	PS
46	Sukumaran	72	M	1.4	NS
47	Devaki	57	F	0.98	NS
48	Lakshmi	64	F	1.08	PS
49	Hemalatha	66	F	1.12	NS
50	Indrani	57	F	1.24	NS
51	Sukumaran	65	M	0.92	NS
52	Krishnan Nair	58	M	0.98	NS
53	Shylaja	44	F	1.24	NS
54	Krishnammal	76	F	0.8	NS

55	Rasathiammal	62	F	1.38	NS
56	Geetha	55	F	1.04	NS
57	Poovathal	69	F	1.08	NS
58	Devaki	61	F	1.03	PS
59	Kavitha	39	F	1.12	NS
60	Nagaraj	68	M	0.98	NS
61	Ruckmaniammal	65	F	1.28	NS
62	Palaniammal	74	F	1.36	NS
63	Savithridevi	62	F	0.98	NS
64	Murugan	67	M	1.02	NS
65	Nirmala	65	F	0.96	PS
66	Rajeswari	56	F	1.21	PS
67	Rangaraj	71	M	0.94	NS
68	Sukumari	64	F	1.01	PS
69	Kaliyammal	69	F	1.04	PS
70	Ramasamy	54	M	1.14	NS
71	Pappammal	63	F	1.08	NS
72	Jeeva	57	F	0.98	PS
73	Rathinabai	51	F	0.94	PS
74	Rangagounder	70	M	1.04	NS
75	Fathima	63	F	1.36	NS
76	Nancy	42	F	1.34	PS
77	Narayanasamy	68	M	1.06	NS
78	Govindammal	64	F	0.94	PS
79	Murali	60	M	0.96	NS
80	Janakaraj	50	M	1.33	NS
81	Rathinbai	69	F	1.24	NS
82	Savithri	70	F	1.25	PS

83	Sakthivel	39	M	1.28	NS
84	Rani	67	F	1.14	NS
85	Shanthi	74	F	0.98	PS
86	Kanagaraj	60	M	1.06	NS
87	Rosy	66	F	0.98	PS
88	Rangasamy	58	M	0.95	NS
89	Sivanandan	45	M	1.08	NS
90	Nesammal	53	F	1.36	NS
91	Thiyagu	71	M	1.14	NS
92	Vasantha	62	F	1.28	NS
93	Divagar	56	M	1	NS
94	Pandian	68	M	0.79	NS
95	Selvi	60	F	1.28	NS
96	Muthupandi	64	M	0.94	NS
97	Rajakumari	70	F	0.98	PS
98	Jackuline	43	F	1.28	PS
99	Janakiammal	99	F	0.81	NS
100	Peterraj	64	M	1.1	NS